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SYMPOSIUM ON THE MECHANISM AND MANAGEMENT OF MENTAL DEFICIENCY IN INFANTS AND CHILDREN

PART I

GUEST EDITOR—I. NEWTON KUGELMASS, M.D., PH.D., SC.D.

Introduction — I. NEWTON KUGELMASS

Genetic Factors in the Etiology of Mental Retardation — HAROLD O. GOODMAN AND C. NASH HERNDON

Recognition of Mental Deficiency in Infancy — OLGA BRIDGMAN

Mental Subnormality: Illusions and Directions — THEBON ALEXANDER

Victor Robinson: A Romantic Medical Historian — GEORGE ROSEN

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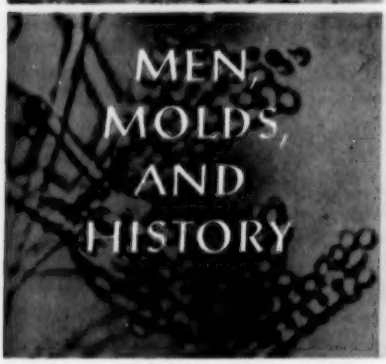

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Symposium on the Mechanism and Management of Mental Deficiency in Infants and Children

Part I

GUEST EDITOR—I. NEWTON KUGELMASS, M.D., Ph.D., Sc.D.

Introduction

I. Newton Kugelmass, M.D., Ph.D., Sc.D.

CONSULTANT, DEPARTMENT OF HEALTH AND HOSPITALS, NEW YORK CITY

NEW YORK, N. Y.

Mental growth is a composite of several fields of behavior, especially motor, adaptive, language, and social behavior, reflected by nutritional and integrative processes occurring in the central nervous system. Adaptive behavior, in particular, reveals the caliber of inherent capabilities embodied in the concept of intelligence. Intelligence tests measure limited functions; developmental tests, all of the child's behavior. Mental retardation is thus a manifestation of cerebral dysfunction. A child's inherent ability to adapt to his environment according to age is delimited by incomplete or impaired mental development arising from prenatal genetic disorders or natal and postnatal disease and injury. The problems of the child ament are always the same, but the solutions differ with each individual in each era. The elimination of the ament by destructive means in ancient times gradually gave way to his exclusion to institutions by obstructive means in medieval times and finally to social adaptation by constructive means in modern times.

Recent discoveries of hereditary biochemical defects in specific enzymes essential for brain metabolism bring several types of amentias into the metabolic realm, amenable to chemotherapy if instituted early, before postnatal cerebral damage progresses. Current developments in the management of Rh factor incompatibility, cretinism, meningitis, syphilis, and other systemic diseases make possible the arrest of serious brain damage with resultant mental defect, provided early treatment is instituted before the brain has been affected irreversibly. Chemical advances in the preparation of newer compounds for the control of behavior disorders are particularly

Part II of this Symposium will appear in the March issue; part III, in the April issue.

beneficial to mentally retarded children because the site of action is on the hypothalamus where the two divisions of the autonomic nervous system integrate, thus affecting deep-seated brain centers rather than the cortex. Surgical progress is limited because severe mental defect is usually due to the absence of normal tissue rather than to the presence of some deleterious focus in the brain; nevertheless, early surgical intervention is effective in craniostenosis and subdural hematoma. The authors of this symposium are leaders in the field with pertinent communications on the mechanism and management of mental deficiency in children.

Beam of Protons Halts Cell Growth of Brain

A narrow beam of high-energy protons, the cores of hydrogen atoms, can be used to injure or destroy cells in specific, limited regions of the brain or central nervous system.

This finding, so far applicable only to animals, could mean that proton beams can halt the growth of cancers or other unwanted growths of the central nervous system in man in the future. Studies of long-term effects of such irradiation on the brain are in progress, six Swedish scientists reported in the British scientific journal *Nature* (November 1.)

The advantage of the narrow proton beam is that it produces sharply defined and limited lesions at the desired spot, with little or no effect on surrounding tissues. The observations apply only to lesions in their relatively early stages, the scientists stressed.

The six are Drs. Borje Larsson, Lars Leksell, Bror Rexed, Patrick Sourander, William Mair, and Bengt Andersson of the Gustaf Werner Institute of Nuclear Chemistry and the Institute of Anatomy, University of Upsala.

Genetic Factors in the Etiology of Mental Retardation

Harold O. Goodman, Ph.D., and C. Nash Herndon, M.D.

DEPARTMENT OF PREVENTIVE MEDICINE AND GENETICS,
BOWMAN GRAY SCHOOL OF MEDICINE OF WAKE FOREST COLLEGE
WINSTON-SALEM, N. C.

Genetic factors play a major role in the causation of many types of mental retardation and are contributory agents in many others. An understanding of the hereditary mechanisms involved in these conditions is clinically valuable for three reasons: First, genetic knowledge provides predictive figures of the risk of recurrence of mental retardation in future children of a couple who have 1 retarded child. This is obviously a matter of intense interest to the parents of a child with any disabling condition. Second, genetic information can aid in the diagnosis of certain clinically distinguishable types of mental retardation and generally permits a more accurate prognosis. Third, in those types amenable to therapy, genetic knowledge may permit anticipation and consequent earlier diagnosis and therapy.

Patients with pathological mental retardation are the victims of pathological mutant genes, of environmental insult, or both. In these patients, a pathological condition has prevented the development of normal intelligence even though they are otherwise adequately endowed both genetically and culturally. The figures in table I suggest that the relative proportion of nonpathological and pathological types of mental defect shifts from a preponderance of nonpathological among the morons to a majority of pathological among the idiots. Clinical and genetic studies support the view that a minority of persons in the moron class exhibit findings denoting pathological changes, whereas almost all idiots adequately studied are found to have a pathological basis for their mental retardation.

In the light of the heterogeneity and complexity of mental deficiency, it is not surprising that many different systems of classification have been used for mental retardation. Table II illustrates a system commonly employed by institutions for the mentally deficient and the frequencies within each category. Because only 5 to 10 per cent of defectives in New York State are institutionalized, and because there is a bias favoring the admission of the more severely retarded, the frequencies given probably do not reflect accurately their corresponding frequencies in the general population of mental retardates. We have chosen the following major categories, which seem appropriate for our purposes: nonpathological mental deficiency, metabolic diseases producing mental deficiency, mental defect involving skeletal anomalies, mongolism, epiloia, and maternal-fetal serological incompatibilities.

NONPATHOLOGICAL MENTAL DEFICIENCY

In using the term "nonpathological" to describe those patients with mental deficiency but without additional discernible pathology, it is recognized that the abnormal intellectual development of these patients depends ultimately on biochemical

TABLE I

Lower Limits of the Distribution of Intelligence in the General Population—Age Group 10–14 Years. Frequencies Given Are Number of Defectives per 10,000 Population (after Penrose[22])

Class	Intelligence quotient range	Normal gaussian distribution prediction	Observed defective persons
Moron	50–70	223	226
Imbecile	20–49	4	24
Idiot	0–19	less than 1	6
Total		227	256

and/or submicroscopic anatomical differences that set them apart from normal individuals. Most patients represent segregation at the low end of the distribution of intelligence quotients in the general population. The fact that the absolute number of undifferentiated patients in table II is larger than the number in the familial class agrees with predictions based on the hypothesis that intelligence is a polygenic trait. Few persons would possess genes leading to the development of either high or low intelligence exclusively, and most would have about the average number. Parents who are mentally retarded have a higher proportion of genes for lower intelligence than do normal parents. Hence, a higher proportion of the children of retarded parents are expected and observed to be retarded. However, the absolute number of retarded parents in the general population is much smaller than the number of persons of average intelligence. Thus, in table II, the proportion of patients classified as undifferentiated exceeds the proportion classified as familial. Most of the genes for lower intelligence in the population as a whole, then, must be found in persons who are of average or normal intelligence. Table III (from Böök,[5] after Brugger, 1939) provides data supporting such a view. All the propositi (index cases) were patients with undifferentiated (nonpathological) mental deficiency. The

TABLE II

Admissions to New York State Schools from April 1, 1947, to March 31, 1955 (Allen and Baroff[1])

Clinical classification	Admissions	
	Number	Per cent
Undifferentiated	4611	38.2
Familial	2986	24.7
Mongolism	1177	9.8
Cranial anomaly	741	6.2
Cerebral palsy	596	4.9
Postinfectional	414	3.4
Post-traumatic	797	6.6
Miscellaneous	744	6.2
Total	12,066	100.0

proportion of siblings of the propositi who are also mentally retarded increases as one progresses from matings in which both parents were normal to matings in which both were mentally defective. Of course, social inheritance is also involved, and again, it is not possible to assess in any precise way the relative contributions of genetic and environmental factors.

What, then, can be said to parents regarding the possible outcome of future pregnancies? Obviously, the risk is not likely to be smaller than the 2 to 3 per cent frequency of mental retardation in the general population. In the case of rare recessive genes, the risk might be as high as 25 per cent. A popular assumption in cases involving environmental insults is that such insults or agents are randomly distributed. However, to assume a random distribution of any agent prior to its identification seems unwise. If an unidentified environmental agent were associated with the occupational or household environment of either parent, this exposure might readily recur during subsequent pregnancies. One might employ figures from table III empirically, but these are institutional figures and may not be representative of the general population. Hence, a reasonable answer to queries regarding future risks would be that they are higher than the 2 to 3 per cent frequency of mental retardation in the general population by an undetermined amount, but with a 25 per cent ceiling for genetic causes. It is conceivable, but quite unlikely, that an environmental causal factor producing a risk greater than 25 per cent would have escaped detection.

METABOLIC DISEASES PRODUCING MENTAL DEFICIENCY

Phenylketonuria. Phenylketonuria (phenylpyruvic oligophrenia) is a metabolic defect characterized by inability to transform phenylalanine into tyrosine, resulting in excretion of phenylpyruvic acid in the urine. Patients with phenylketonuria exhibit mental retardation of severe degree, about 60 per cent being at the idiot level. Extrapyramidal signs are frequent, including muscular rigidity, hyperreflexia, and athetoid movements. Stature is often reduced and the head measurements small. More than two thirds of affected persons are blondes with blue eyes, with skin sensitive to sunlight. Crow and Schull[13] have interpreted this as evidence of a

TABLE III

Incidence of Mental Deficiency among the Siblings of Propositi with "Mental Deficiency of Unknown Etiology" (Book,[5] after Brugger, 1939)

Author	Both parents normal		One parent defective		Both parents defective	
	Number of siblings	Per cent defective	Number of siblings	Per cent defective	Number of siblings	Per cent defective
Lokay	123	13.0	36	33.0	5	100.0
Brugger	310	17.8	124	41.3	41	93.2
Wildenskov	94	13.8	72	40.3	78	93.6
Kreyenberg	753	15.9	280	33.9	23	82.5
Frede	278	17.3	82	48.8	81	90.1

quantitative reduction in pigment formation secondary to a reduced supply of tyrosine.

Genetic analysis of several groups of families indicates that affected children are homozygous for (have a double dose of) a recessive gene. Several other rare types of mental deficiency are also governed by recessive genes, and what will be said of the genetics of phenylketonuria applies as well to them.

The probability that parents who have produced one phenylketonuric child will have another with the same condition is the familiar 1 in 4 ratio, or 25 per cent, and remains so for each and every subsequent pregnancy. Two out of 3 of the normal siblings will be carriers. Normal siblings are not likely to have affected children because their chance of marrying a carrier is slight (about 1 in 100 in the general population is a carrier of the gene for phenylketonuria).[24] Marriage of a normal sibling to a cousin is potentially dangerous, however, as the same recessive gene would be found in a much larger proportion of relatives than in unrelated persons.

Hsia et al[22] attempted to identify persons heterozygous for the gene for phenylketonuria by means of a phenylalanine tolerance test. With slight overlap, they were able to distinguish carriers from homozygous normal persons. Berry et al[1] confirmed the value of the test. Knox and Messinger[25] found that the fasting plasma phenylalanine level was also elevated in presumptive heterozygotes (parents and two thirds of normal siblings of phenylketonuric patients). They found that the use of the phenylalanine tolerance test coupled with a basal plasma phenylalanine test would identify a greater number of heterozygotes than would either test alone. Thus, at the biochemical level, the gene for phenylketonuria is not a recessive gene, but an incompletely dominant one, i.e., a gene that expresses itself to some extent in the heterozygote but to a greater extent in the homozygote.

Horner and Streamer[21] reported that a boy diagnosed at 7 weeks of age as a phenylketonuric and begun on a phenylalanine-restricted diet at the same time appeared to show no signs of mental retardation at 9 months. It is of incidental interest that the disease in that instance was anticipated from study of an older affected sibling. The child's urine was negative by the ferric chloride test at birth, suggestively positive at 5 weeks, and unequivocally positive at 7 weeks. The measure of amenability to therapy, then, is not whether genetic factors are involved in a disease, but whether available therapy is sufficiently specific.

Niemann-Pick Disease. Niemann-Pick disease is characterized by mental deterioration and deficiency, but patients are not usually institutionalized because the condition is rapidly fatal. Our reason for including it here is its possible relationship to infantile amaurotic idiocy. Niemann-Pick disease is an intracellular metabolic defect, with retention of sphingomyelin, a diaminophosphatide, primarily in histiocytes and reticular cells, and also involving the ganglion cells of the brain. There is widespread accumulation of characteristic large, pale foam cells (Niemann-Pick cells). The disease usually begins in infancy with loss of weight, dehydration, hepatosplenomegaly, and emaciation. Lymphadenopathy and skin pigmentation are common. A progressive paralysis without reflex changes appears, and about 60 per cent of patients develop a cherry red to reddish brown spot at the macula indistinguishable from that seen in infantile amaurotic idiocy. Mental deterioration

is rapid, and about three fourths of the patients die before the second birthday. Videback[44] has summarized clinical and genetic data on 73 cases.

Approximately half of the reported cases of Niemann-Pick disease mention Jewish ancestry of the patients. The condition is equally common in males and females. About one fourth of the reported cases describe one or more affected siblings, and the disease has not been found in more than one generation of a family. It seems likely that the condition is due to an autosomal (not sex-linked) recessive gene, homozygotes being affected and heterozygotes apparently normal.

Amaurotic Idiocy. The terms "amaurotic idiocy" and "cerebromacular degeneration" are applied to a small group of closely related conditions characterized by abnormal lipid deposits in the nervous system. The clinical varieties usually described, with their characteristic ages of onset, are as follows: (1) infantile amaurotic idiocy (Tay-Sachs disease), less than 1 year; (2) late infantile type (Bielschowsky disease), 2 to 4 years; (3) juvenile amaurotic idiocy (Spielmeyer-Vogt disease), 3 to 10 years; (4) late juvenile type (Kufs disease), 15 to 25 years.

Infantile amaurotic idiocy (Tay-Sachs disease) is characterized by early onset and relatively rapid progression, with hypertonicity, listlessness, progressive spastic paralysis, blindness, convulsions, bulbar palsy, and death by the third year. A cherry red spot at the macula is classical, but is sometimes absent. Juvenile amaurotic idiocy (Spielmeyer-Vogt disease) is similar but the onset is later and the course slower. Blindness and convulsions are prominent in the early stages, with mental deterioration, which progresses to amentia. Death occurs within 10 to 15 years. The macular lesion is more diffuse and brownish than that seen in the infantile type. The late infantile type or Bielschowsky disease is quite rare and is intermediate in course and symptomatology between the Tay-Sachs and Spielmeyer-Vogt types. The late juvenile variety or Kufs disease is milder and is usually without blindness. A detailed summary and comparison of the clinical features in the four age groups were given by Rothstein and Welt,[40] and the pathological features were described by Ford.[15]

A careful genetic study of juvenile amaurotic idiocy was made by Sjögren,[41] demonstrating that the disease is due to an autosomal recessive gene. Among 145 sibships containing affected children, the parents were first cousins in 23 (16 per cent) and related in other ways in 26 (18 per cent). Bagh and Hortling[3] noted that vacuolization of the lymphocytes was a constant finding in juvenile amaurotic idiocy, and Rayner[36] found the same abnormality in both parents and in 6 of 7 normal siblings of 2 unrelated patients. Rayner suggested that genetic carriers of the recessive gene may be identified by the presence of vacuolated lymphocytes.

The two types, infantile and juvenile, have not been known to occur in the same family. The transitional types described by Bielschowsky and Kufs have not been adequately studied by genetic methods. The possibility that Tay-Sachs disease and Niemann-Pick disease may be merely variants of one basic disease has been suggested, largely on the basis of marked clinical and pathological similarities. The argument for identity of the two conditions is strengthened by the observation that typical cases of Tay-Sachs disease and Niemann-Pick disease have been found in a single sibship.[43] The question of the possible genetic identity of the two diseases

must remain open, but fortunately has no real bearing on treatment or on advice to be given to a specific family. As the autosomal recessive mechanism applies to both conditions, after 1 affected child has appeared in a sibship, the probability that a child born later will develop the condition is 25 per cent.

Cretinism. Endemic cretinism has been studied genetically by several investigators. The high frequency of cretinous offspring of goitrous mothers and the fall in the frequency of cretinism among offspring when mothers of cretins had left the endemic area led some investigators to conclude that heredity plays no part in its etiology. However, the genetic study of a trait as common as cretinism in endemic areas presents special problems. For example, absence of an excess of consanguineous unions is not evidence against recessive inheritance when the frequency of the defect exceeds the frequency of cousin marriages in the population as a whole. The interaction between maternal and fetal metabolism further complicates the identification of genetic factors. But the fact that all persons on iodine-deficient diets are not goitrous or cretinous suggests that susceptibility or resistance to the effects of low iodine intake may reflect inherent constitutional differences.

Sporadic goitrous cretinism is only 5 to 10 per cent as common as the nongoitrous type, and the relationship between the two types is presently unknown. Several recent excellent studies have shed light on the complexities of congenital goitrous cretinism.[23, 33, 42] Stanbury and McGirr[42] stated that there may be as many as three genetically and biochemically distinct defects that can lead to the same clinical condition, their similarity resulting from the fact that all three lead ultimately to insufficient thyroid hormone production. They suggested that a series of genes affect the production of a series of enzymes, which, in turn, regulate the various steps in the synthesis of thyroid hormone. Defects in the synthetic chain described thus far are: (1) deficiency of an oxidative enzyme resulting in deficient utilization of trapped iodide in the thyroid gland; (2) deficiency in the rate of coupling iodotyrosines with iodothyronine; (3) deficiency of dehalogenase permitting escape and excretion of monoiodotyrosine and diiodotyrosine.[42] Stanbury and McGirr postulated that the genetic bases for these three defects are three different autosomal recessive genes. Support for this concept is provided by the fact that parents of affected children are clinically normal, by the observed familial occurrence, and by the excess of consanguineous marriages among parents of affected children. Within each family, secondary cases invariably appear to have the same defect as the index cases. Clinical and biochemical study of the effects of uncommon recessive genes has added greatly to our knowledge of normal metabolic pathways in thyroid hormone synthesis.

Galactosemia is a congenital metabolic defect based on inability to convert dietary galactose into glycogen, secondary to enzyme deficiency. Affected infants subsisting on a milk diet are thus relatively starved and develop widespread tissue damage. There is marked retardation of growth and mental development, often with hepatomegaly, cataract formation, and digestive disturbances. If affected infants can be placed on a lactose-free diet before permanent tissue damage occurs, physical and mental development will proceed in a normal manner. Genetic studies strongly suggest that affected infants are homozygous for an autosomal recessive gene. Holzel,

Komrower, and Schwarz[20] pointed out that abnormal galactose tolerance tests are observed in certain normal parents and siblings of patients with galactosemia, showing that the carrier or heterozygous state of the gene can often be identified. However, they found some families in which only 1 parent presented a detectable biochemical abnormality, suggesting either that the test does not detect all carriers or that more than one mechanism of inheritance may be involved. In families where 1 case of galactosemia has appeared, the probability that younger siblings may exhibit the disease is approximately 25 per cent.

Gaucher's disease is an uncommon disorder of lipid metabolism characterized by the accumulation of the cerebroside kerasin in the cells of the reticuloendothelial system. The resulting hyperplasia leads to enlargement of the spleen, liver, and lymph nodes. In cases with very early onset, the brain may be involved with resulting mental retardation. The genetic basis for Gaucher's disease is an autosomal recessive gene.[18]

The Laurence-Moon-Biedl syndrome is a disease in which one can presume there is a fundamental metabolic dysfunction, but despite some knowledge of its genetic etiology, the primary physiological defect has yet to be revealed. The components of the syndrome are hypogenitalism, retinitis pigmentosa, obesity, mental deficiency, and polydactylism, but any patient may lack one or more of these signs. The mental age of most patients is 5 to 7 years.[32] There is consanguinity of the parents of about one third of the affected children.[14] It seems likely that affected persons are homozygous for an autosomal recessive gene, but this hypothesis does not adequately account for the occasional appearance of minor forms of the syndrome in more than 1 sibship of certain families.

MENTAL DEFICIENCY WITH SKELETAL ANOMALIES

Skeletal anomalies, particularly those involving the central nervous system, comprise a large proportion of pathological mental deficiency. A majority of these anomalies are congenital and are associated with severe mental retardation, though with some defects patients occasionally have normal intelligence. The studies of Murphy[28] and of Record and McKeown[37–39] provide the best data available on population frequencies and risks of recurrence of congenital malformations of the central nervous system.

Microcephaly. Microcephaly is a difficult condition to define. The use of head size as a criterion (classically, a circumference of less than 13 inches in the adult) may include some persons with other recognized diseases, such as mongolism. Böök et al[7] suggested the designation microcephaly when the circumference of the patient's skull is three standard deviations below the mean for the respective age and the condition is not due to premature synosteoses of the cranial sutures. In addition to the small head size, the shape of the skull is often abnormal with a marked recession of the forehead and a flattened occiput. Weight and height of the microcephalic person are generally clearly below the average. Neurological signs are not diagnostic.

Microcephaly is of special interest because there is evidence for multiple etiology, which is as convincing as retrospective studies can provide. Böök et al,[7] in sum-

marizing etiological bases, included maternal pelvic irradiation, prenatal or neonatal infections, especially maternal rubella, and birth trauma or asphyxia as three reasonably well-documented causes of microcephaly. Plummer's[24] findings in a study of 205 infants who had been exposed in utero during the first 20 weeks of gestation to the atomic bomb at Hiroshima are particularly striking. Of 11 exposed within 1200 meters of the hypocenter, 7 were born microcephalic. There were no cases of microcephaly among 194 exposed at greater distances. Murphy[28] studied 75 infants born at term of mothers who had received therapeutic doses of pelvic irradiation during pregnancy. Thirty-eight of these infants were abnormal, 16 of them being microcephalic. It seems clear that exposure to radiation during early pregnancy may cause brain damage that results in the clinical picture of microcephaly. It is also well established that microcephaly is one of the malformation types that may be caused by rubella during the first trimester of pregnancy.[27] Anoxia of the fetus may also be an effective environmental insult capable of producing the clinical picture of microcephaly. These environmental agents are nonspecific and probably act by depressing brain growth at a critical stage in embryogenesis.

Microcephaly that is clinically indistinguishable from the environmentally induced types occurs as a result of gene action. Böök et al[7] and Komai et al[26] have studied the families of patients with microcephaly whose condition could not be attributed to one of the environmental agents mentioned previously. They obtained excellent evidence to show that these patients were homozygous for an autosomal recessive gene for microcephaly. Böök et al estimated that the population frequency of patients with the inherited type of microcephaly lies between 1 in 25,000 and 1 in 50,000.

Advice to the parents of a microcephalic child concerning future pregnancies can be a bit difficult. A careful obstetric history may identify an etiologically significant environmental factor in some families. In this case, future children would not be likely to have microcephaly if the mother can be protected from the identified environmental agent. In most families no such agent will be apparent. It seems wise to presume that any given case of microcephaly is of genetic etiology until proved otherwise. The recurrence risk of 25 per cent will be found to hold true for the majority of families.

Acrocephaly. Increased skull height relative to length and breadth is common to acrocephaly, scaphocephaly, and oxycephaly. In acrocephaly and scaphocephaly the skull is flattened and is elongated anteroposteriorly, whereas in oxycephaly the skull tapers from all sides. In the classic type of acrocephaly, Crouzon's disease or craniofacial dysostosis, there is a central prominence of the frontal region of the skull, a beaklike nose, exophthalmos, exotropia, and prognathism. Mentality is often normal, and when mental defect is present, it is usually not severe. Falls[14] reviewed reported cases and concluded that the disease may be transmitted by a dominant gene with fairly high penetrance, i.e., the gene produces the expected trait in a high proportion, but not in all persons who receive it. Sporadic cases of the disorder may result from new mutations. Any prediction of future risk should be based on a searching examination of the family history. If prior cases of the disorder have occurred in the aunts, uncles, or grandparents, probabilities heavily favor

the assumption that the normal parent in whose family prior cases occurred has received and transmitted the gene, but has not developed clinically apparent abnormality.

Human genes, both dominant and recessive, showing some degree of incomplete penetrance, are the rule rather than the exception. Both environmental and genetic factors may explain the phenomenon, but the specific factors responsible for lack of penetrance are unknown in most instances. The parent who is heterozygous for a rare dominant gene has, of course, the usual 50 per cent chance of transmitting the potentiality, but the probability that a child of such a parent will develop that trait should be reduced by the percentage of penetrance shown by that gene. Unfortunately, the number of rare genes for which accurate penetrance figures are available is extremely small.

In summary, a sporadic case of a rare dominant trait is most likely either a new mutation or the manifestation of a gene showing incomplete penetrance. The more severe the disease, the more likely a sporadic case is of mutational origin. Because the probability of occurrence of a second independent mutation is very small, birth of 2 affected persons in a single sibship greatly weakens the mutational hypothesis and suggests alternative explanations, including recessive inheritance.

Acrocephalosyndactyly. Acrocephalosyndactyly (Apert's syndrome) is characterized by a towering skull flattened anteroposteriorly, syndactyly of the fingers and toes, and hypertelorism associated with a broadened forehead. The disease involves the whole skeletal system to a lesser degree, and mental retardation is common.[32] Inheritance is frequently regulated by a dominant gene, but some pedigrees suggest recessive inheritance.[14] Although about 100 cases are recorded in the literature, only 15 report transmission from one generation to the next, the remainder being sporadic cases.[6]

Arhinencephaly and Rhinencephaly. Our meager knowledge of arhinencephaly and rhinencephaly stems from their extreme rarity. The term "arhinencephaly" is applied to conditions in which the olfactory bulb and tracts and some immediately associated structures are absent.[35] Total absence of these structures has been found without other anomalies of the head, and a great part of the rhinencephalon is regularly absent in cyclopia. However, in cases described by Grebe,[17] the patients were mentally retarded and had associated defects including median cleft palate, hypoplasia of the nasal septum, and absence of olfactory sense.

Rhinencephaly observed by 1 of the present authors in 2 unrelated children included median cleft palate, nasal mucosa occupying the upper right quadrant of the face, and mental retardation. The 2 children differed in that in 1 the nasal mucosa was covered by normal skin, whereas in the other the nasal mucosa was completely exposed. Grebe observed several cases in the same family and suggested that a dominant gene with variable manifestation was a likely explanation since other relatives appeared to be slightly affected.

Hypertelorism. Ocular hypertelorism (abnormally increased distance between the eyes) is observed as a part of several syndromes, but may occur without other cranial anomalies. It is often associated with mental retardation. Hypertelorism has frequently been observed in more than 1 member of a sibship, and also may occur in

parent and child. Some pedigrees in the literature suggest the action of a dominant gene, while others are more suggestive of recessive inheritance. A critical genetic study of this condition has not been done. It seems obvious that there is a very real risk of occurrence of this defect among either the siblings or children of a known case, but the magnitude of this risk cannot yet be specified.

Gargoylism (Lipochondrodystrophy). Gargoylism, also known as the Hunter-Hurler syndrome, is basically a metabolic defect. It is usually classified with the varieties of mental retardation with skeletal anomalies because of the diagnostic importance of the bone and joint changes. The pathological changes include intracellular storage of abnormal lipids, but Brante[10] has shown that the lipid deposits are secondary to a defect in the metabolism of mucopolysaccharides. Gargoylism is characterized by mental deficiency, hepatosplenomegaly, corneal clouding, and multiple bony deformities, which result in dwarfing, limited joint extension, skull deformities, and characteristic radiological findings.

In an analysis of 213 cases of gargoylism in 150 families,[19] it was shown that two genetic varieties exist. Among cases reported in the literature, the affected children are homozygous for an autosomal recessive gene in about 65 per cent of families, while a sex-linked recessive gene is responsible in about 35 per cent of families. It was found that the recessive and sex-linked types of gargoylism may usually be distinguished clinically. In the sex-linked type, corneal clouding is absent, dwarfing is less frequent (33 per cent), but nerve deafness is common (43 per cent). In the autosomal recessive type, corneal clouding and dwarfing are common (81 per cent and 73 per cent, respectively), while deafness is rare (5 per cent). Differentiation of the genetic types is of particular significance with regard to advice on marriage for normal sisters of gargoyls.

MONGOLISM

Mongolism is one of the most frequent types of pathological mental deficiency. Böök and Reed[5] estimated the population frequency to be between 1 in 500 and 1 in 1500, and Øster[29] estimated its frequency in Denmark as 1 in 618 live births. The diagnosis of mongolism is based on the occurrence of a constellation of traits, no one of which is a prerequisite to the diagnosis nor limited to the population of mongoloids. Penrose[32] set up criteria for diagnosis using the following seven stigmas: Binet intelligence quotient from 15 to 29, cephalic index 0.83 or higher, epicanthic fold over either eye, fissured tongue, presence of blepharitis, transverse fold on either palm, single flexion crease on either little finger. He stated that any patient with four or more of these characteristics is almost certain to be a mongoloid imbecile. Walker[45] has developed an objective method of diagnosing mongolism from dermatoglyphic analyses, which, in her material, correctly identified 70 per cent of a group of known mongoloids.

Many etiological hypotheses have been proposed to account for mongolism, and no single hypothesis, as yet, fits all the observations. Unfortunately, many of those suggested are presently untestable. Several studies have shown clearly the increased risk of bearing mongoloid offspring associated with increasing maternal age.[11, 29, 31] It has also been shown that the frequency of this defect is largely in-

dependent of birth rank, parity, and paternal age. Some have interpreted the maternal age effect as excluding the presence of genetic factors in mongolism. However, although providing evidence of an environmental component, a relationship between maternal age and the frequency of mongolism is evidence neither for nor against a genetic hypothesis. Twin studies in mongolism have shown high concordance among identical twin pairs and low concordance among fraternal pairs. However, pedigree analyses have not revealed the mode of inheritance of the genetic factor(s) involved. Øster's[30] study of the familial occurrence of mongolism presented a surprising paradox. One would predict on the basis of the well-demonstrated maternal age effect that if multiple cases of mongolism were to occur in a sibship, they would occur among the offspring of older mothers. However, as is shown in table IV, all multiple cases occurred in families in which the mother was less than 35 years of age. Using the Poisson distribution, statistical analysis indicates that there is a highly significant excess of mongols over expected numbers born to mothers 35 years of age or younger whether one considers all siblings of mongoloids or only those born subsequent to the index case. Confirmation of this observation in similarly unselected data would discourage the use for counseling of previously calculated empirical risk figures[5] for mongolism.

EPILOIA (TUBEROUS SCLEROSIS, BOURNEVILLE'S DISEASE)

Epiloia is an uncommon disease characterized by sebaceous adenomas, convulsive seizures, and mental defect. The skin is involved in 60 to 70 per cent of the cases,[9] and a butterfly rash (sebaceous adenomas about the nose) is characteristic. Subungual fibromatoses often arise at puberty. Café au lait spots and pigmented, verrucous, or anemic nevi have often been reported. Epileptiform seizures occur in about 80 per cent of cases and may be of either the focal or the generalized type. The clinical signs do not correlate well with pathological cerebral changes. Mental deficiency occurs in about 70 per cent of the cases and intelligence test quotient scores range from borderline normal to the idiot level. The lesions presumably responsible

TABLE IV

Mongolism Observed and Expected among the Siblings of Mongoloids Born after and among Those Born both before and after the Propositus. Distributed According to Age of Mother at Time of Birth of Siblings (after Øster[30])

Age of mother	Siblings born after propositus			Siblings born both before and after propositus		
	Number of siblings	Mongoloids		Number of siblings	Mongoloids	
		Observed	Expected		Observed	Expected
15–34	166	4	0.149	1392	7	1.162
35–49	177	0	1.260	414	0	2.324
Total	343	4	1.409	1806	7	3.486

for both the epilepsy and mental retardation are sclerosed areas of tumor formation in the brain.

Borberg[9] found positive family histories in 42 per cent of his propositi, the remainder being sporadic cases. In his material, the genetic factor appeared to be an irregular dominant gene. Other studies showed that the clinical manifestation of the gene may be highly variable.[32]

MATERNAL-FETAL SEROLOGICAL INCOMPATIBILITY

Erythroblastosis fetalis is an uncommon cause of mental deficiency, but there is little doubt that patients with severe brain damage may be mentally retarded. Differences in the Rh system of genes are responsible for most maternal-fetal incompatibility of clinical significance. The retardation is presumably the result of nonspecific injury to the brain by the products of hemolysis or prolonged anoxia.

Crome and associates[12] pointed out some of the difficulties in diagnosing mental deficiency due to Rh incompatibility. They described the triad, bilateral athetosis, deafness, and mental retardation, as comprising the "typical" syndrome of erythroblastotic brain damage. However, they recognized that only a minority of cases present the complete pattern. In all cases there was a history of severe neonatal jaundice, mental retardation, and various kinds and degrees of motor defect.

Patients who survive neonatal jaundice without residual motor defects usually have been presumed to develop into completely normal persons. However, Gerver and Day[16] studied the intelligence quotient test scores of 68 "normal" children free of motor defects who had survived erythroblastosis fetalis sufficiently severe to have required transfusions and who had had serological evidence of maternal-fetal incompatibility. The average age of their index cases was 4.5 years. They found that the mean intelligence quotient score of the "normal" children was 11.8 points below the mean score of their older nonaffected siblings. They also found that the mean score of the children who had recovered from erythroblastosis was significantly lower than the mean score of an age-matched control series. The distribution of scores of recovered children was unimodal, a fact that indicates that the lower mean score of the affected group did not result from averaging the scores of a few severely defective children with those of a larger nondefective group. Hence, although severe mental defect is uncommon among survivors of erythroblastosis fetalis, a moderate reduction in intelligence quotient is not unlikely.

SUMMARY

This discussion has attempted to evaluate current concepts of the role played by genetic factors in the etiology of mental retardation. It has been emphasized that in a large proportion of cases, borderline and moron intelligence levels are not pathological and represent the chance accumulation of normal genetic factors determining low-grade intellectual ability. Pathological mental retardation results whenever etiological agents, which may be environmental, genetic, or both, suppress the development of intelligence in persons with otherwise normal genetic and cultural endowments. Specific types of pathological mental retardation can be separated from

the etiologically heterogeneous group of "undifferentiated" mental defect only if there is some associated physical or biochemical abnormality. The clinically recognized types in which genetic factors are etiologically prominent were discussed. Emphasis has been placed on the usefulness of an appreciation of the role of genetic factors as an aid to early diagnosis and therapy and in providing risk estimates for recurrence of mental defect within family groups.

BIBLIOGRAPHY

1. Allen, G., and Baroff, G. S.: Mongoloid twins and their siblings, Acta genet. 5:294–326, 1955.
2. Allen, G., and Kallmann, F. J.: Frequency and types of mental retardation in twins, Am. J. Human Genet. 7:15–20, 1955.
3. Bagh, K. V., and Hortling, H.: Blodfynd vid juvenil amaurotisk idioti, Nord. med. 38:1072–1076, 1948.
4. Berry, H.; Sutherland, B., and Guest, G. M.: Phenylalanine tolerance tests on relatives of phenylketonuric children, Am. J. Human Genet. 9:310-316, 1957.
5. Böök, J. A., and Reed, S. C.: Empiric risk figures in mongolism, J.A.M.A. 143:730–732, 1950.
6. Böök, J. A., and Hesselvik, L.: Acrocephalosyndactyly, Acta paediat. 42:359–364, 1952.
7. Böök, J. A.; Schut, J. W., and Reed, S. C.: A clinical and genetical study of microcephaly, Am. J. Ment. Deficiency 57:637–660, 1953.
8. Böök, J. A.: Oligophrenia. *In:* Sorsby, A., editor: Clinical Genetics, St. Louis, C. V. Mosby Co., 1953, 603 pp.
9. Borberg, A.: Clinical and genetic investigations into tuberous sclerosis and Recklinghausen's neurofibromatosis, Op. Dom. Biol. Hum. Univ. Hafn. 23:239, 1951.
10. Brante, G.: Gargoylism: a mucopolysaccharidosis, Scandinav. J. Clin. & Lab. Invest. 4:43–46, 1952.
11. Carter, C., and MacCarthy, D.: Incidence of mongolism and its diagnosis in the newborn, Brit. J. Soc. Med. 5:83–90, 1951.
12. Crome, L.; Kirman, B. H., and Marrs, M.: Rhesus incompatibility and mental deficiency, Brain 78:514–536, 1955.
13. Crow, F. W., and Schull, W. J.: Phenylketonuria: studies in pigment formation, Fol. Hered. Path. 1:259–268, 1952.
14. Falls, H. F.: Skeletal system, including joints. *In:* Sorsby, A., editor: Clinical Genetics, St. Louis, C. V. Mosby Co., 1953, 603 pp.
15. Ford, F. R.: Diseases of the Nervous System in Infancy, Childhood, and Adolescence, ed. 2, Springfield, Ill., Charles C Thomas, 1944.
16. Gerver, J. M., and Day, R.: Intelligence quotients of children who have recovered from erythroblastosis fetalis, J. Pediat. 36:342–348, 1950.
17. Grebe, H.: Zur Ätiology der Arhinencephalie, Erbarzt. 12:138, 1944. Cited by Penrose.[32]
18. Herndon, C. N., and Bender, J. R.: Gaucher's disease: cases in five related Negro sibships, Am. J. Human Genet. 2:49–60, 1950.
19. Herndon, C. N.: Genetics of the lipidoses, Research Publ., A. Nerv. & Ment. Dis. 33:239–258, 1954.
20. Holzel, A.; Komrower, G. M., and Schwarz, V.: Galactosemia, Am. J. Med. 22:703–711, 1957.
21. Horner, F. A., and Streamer, C. W.: Effect of a phenylalanine-restricted diet on patients with phenylketonuria, J.A.M.A. 161:1628–1630. 1956.
22. Hsia, D. Y.; Driscoll, K. W.; Troll, W., and Knox, W. E.: Detection by phenylalanine tolerance tests of heterozygous carriers of phenylketonuria, Nature 178:1239–1240, 1956.
23. Hutchison, J. H., and McGirr, E. M.: Sporadic non-endemic goitrous cretinism: hereditary transmission, Lancet 270:1035–1037, 1956.
24. Knox, W. E., and Hsia, D. Y.: Pathogenetic problems in phenylketonuria, Am. J. Med. 22:687–702, 1957.
25. Knox, W. E., and Messinger, E. C.: The detection in the heterozygote of the metabolic effect of the recessive gene for phenylketonuria, Am. J. Human Genet. 10:53–60, 1958

26. Komai, T.; Kishimoto, K., and Ozaki, Y.: Genetic study of microcephaly based on Japanese material, Am. J. Human Genet. *7*:51–65, 1955.
27. Lundström, R.: Rubella during pregnancy, Acta paediat. *41*:583–594, 1952.
28. Murphy, D. P.: Congenital Malformations, ed. 2, Philadelphia, J. B. Lippincott Co., 1947, 127 pp.
29. Øster, J.: Mongolism, Op. Dom. Biol. Hum. Univ. Hafn. *32*:206, 1953.
30. Øster, J.: The causes of mongolism, Danish M. Bull. *3*:158–164, 1956.
31. Penrose, L. S.: A method of separating the relative aetiological effects of birth order and maternal age with special reference to mongolian imbecility, Ann. Eugenics *6*:108, 1934.
32. Penrose, L. S.: The Biology of Mental Defect, London, Sidgwick and Jackson, Ltd., 1949, 285 pp.
33. Pickering, D. E.; Sheline, G. E., and Crane, J. T.: Sporadic familial goitrous hypothyroidism, Am. J. Dis. Child. *93*:510–518, 1957.
34. Plummer, G.: Anomalies occurring in children exposed in utero to the atomic bomb in Hiroshima, Pediatrics *10*:687–693, 1952.
35. Rasmussen, A. T.: Anomalies of the central nervous system. *In:* Baker, A. B., editor: Clinical Neurology, vol. 3, New York, Paul B. Hoeber, Inc., 1955.
36. Rayner, S.: Juvenile amaurotic idiocy, Acta genet. *3*:1–5, 1952.
37. Record, R. G., and McKeown, T.: Congenital malformations of the central nervous system. I. A survey of 930 cases, Brit. J. Soc. Med. *3*:183–219, 1949.
38. Record, R. G., and McKeown, T.: II. Maternal reproductive history and familial incidence, Brit. J. Soc. Med. *4*:26–50, 1950.
39. Record, R. G., and McKeown, T.; III. Risk of malformation in sibs of malformed individuals, Brit. J. Soc. Med. *4*:217–220, 1950.
40. Rothstein, J. L., and Welt, S.: Infantile amaurotic family idiocy, Am. J. Dis. Child. *62*:801–843, 1941.
41. Sjögren, T.: Die juvenile amaurotische Idiotie, Hereditas *14*:197–426, 1931.
42. Stanbury, J. B., and McGirr, E. M.: Sporadic or non-endemic familial cretinism with goiter, Am. J. Med. *22*:712–723, 1957.
43. Van Bogaert, L., and Klein, D.: Observations sur l'hérédité des idioties amaurotiques et de la splénohépatomégalie lipidienne (11 familles), J. génét. humaine *4*:23–78, 1955.
44. Videback, A.: Niemann-Pick's disease: acute and chronic type? Acta paediat. *37*:95–116, 1949.
45. Walker, N. F.: The use of dermal configurations in the diagnosis of mongolism, J. Pediat. *50*:19–26, 1957.

Drug Sales Rise

Drug sales expanded more than sevenfold in less than two decades, according to a *Barron's* (financial weekly) estimate. The publication put 1958 sales at $2.25 billion, compared with $300 million in 1939.

Proprietary drugs accounted for about $550 million or 25 per cent of last year's total, compared with 50 per cent in 1939 before the advent of antibiotics and other new ethical drugs.

The top-selling item among the proprietaries are vitamin concentrates, followed by cough and cold medicines and aspirin compounds; these three groups account for more than half the over-the-counter sales.

Recognition of Mental Deficiency in Infancy

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Mental deficiency is not a separate entity occurring as an isolated symptom in an otherwise healthy person, but is one of the accompaniments of almost innumerable conditions of which mental defect is only a part. In newborn infants there are often variations of behavior, which may be significant as indications of future development, and the mother of a young child will even tell at times of her belief that the infant before birth was different from her other children, that it was inactive and overquiet, or perhaps that it was so violently active that she feared something might be amiss.

The normal newborn infant has already acquired a considerable repertoire of responses, some of which are at a reflex level and do not indicate cortical activity. Crying, sucking, and a reflex grasp can be noted in some newborn children with little cortical tissue, where the possibility of normal mental development with continued growth is quite out of the question. On the other hand, there are some bits of behavior in the healthy newborn infant that seem to indicate a participation of the cortex in the responses. The ability of the infant only a few days old to distinguish between cow's milk and goat's milk has often been reported by mothers, and the definite response of a very young infant if a drop of vinegar is put on the tongue indicates that sensory perception has developed and that an apparent voluntary reaction to unpleasant stimulation is possible. On the other hand, with much older, severe defectives such reactions may be entirely absent. This may be due to sensory deficiency or it may represent an inability to appreciate or evaluate simple stimuli even when the sensory apparatus is intact.

We think of tests of intelligence as being possible only when the child is old enough to talk and to respond to verbal commands, but if it is remembered that mental retardation is one symptom of a general deficiency, we can use even these earliest responses of a very young infant as a tentative measure of its maturity. Normal newborn infants spend most of their time sleeping between feedings, but one baby when awake will be alert and interested, while another for weeks and months seems to disregard its surroundings completely. When the infant never moves unless disturbed, makes no response to bright objects, which most babies of only a few weeks regard with interest, is limp and relaxed when picked up, and makes no response to the mother's voice, there is some reason to believe that all is not well. On the other hand, constant fretfulness, monotonous crying, little sleep, poor feeding, and restless irritability may often be indicators of failure of normal development.

The need for careful examination, study of possible causes, and a consideration of possible prognosis is obvious for any infant whose behavior and rate of development show a marked deviation from the average. Parents who are faced with the evidence that their child is different from other children need a great deal of help. Their first fear is invariably that there may be some hereditary taint, and the search of

their family trees is often pathetic. Some seem consumed with guilt, and many a young, healthy mother whose first baby was not normal will be afraid to have more children even when the chance of having another defective child is extremely small. In some such families, parents have devoted their lives to caring for a single child who can never lead an independent life. It is such cases as these that require the most careful investigation of possible causes, with as full an explanation to the parents as possible. This does not mean that an immediate diagnosis of idiocy should be made, but it does mean that there should be careful and thorough diagnostic investigations and that phases of later development in the child should be noted carefully. A practical method for noting mental and social growth is the Vineland Social Maturity Scale, which serves not only to check the rate of the child's acquisition of simple functions, but is often very useful in helping parents to realize what can ordinarily be expected of the child as time goes by.

Tests of the child's vision and hearing should also be made regularly, since an infant with sensory deficiency cannot be expected to respond as does the normal child. Another problem relates to the child's general health. Defective children are often sickly children and sick babies are never as alert and responsive as healthy ones. The question often arises as to whether the apparent lack of mental alertness is due to illness from which the baby can recover completely or whether the physical frailty is part of a general deficiency associated with mental retardation.

The assumption made so far has been that the infants under consideration are in homes where they have been surrounded by care and affection and where there has been no gross deprivation in the environment. It is true that the great majority of mental defectives, often picturesquely called the "garden variety," come from families where social and often mental inadequacy are common, with parents, brothers, and sisters frequently abnormal, and where the environment has been one of deprivation so far as physical care is concerned and where the lack of affection and stimulation may have been responsible for the child's failure. These are the cases that come to the attention of welfare organizations after serious neglect and where the final capacity of the child cannot be judged until there has been a long period of consistent, affectionate, and stimulating care. At times the results of good care and training are so marked that one is led to conclude that the retardation is environmental, and at other times even years of good care seem to accomplish little. Most of such patients are relatively mild defectives. Many are later found in institutions, but many continue to live in the community, tending to perpetuate the conditions from which they themselves have suffered.

Severe defectives, on the other hand, tend to occur in families as good as or better than the average. The parents and other children in the family are normal, and the defective child is generally strikingly different from the other members of the family.

Investigation of possible causes has for its object cure, if possible, amelioration of the condition where cure cannot be brought about, and finally, the accumulation of information that may lead to prevention.

Fortunately, relatively few of the conditions found are proved to be hereditary. When similar cases have occurred in more than one member of a family, heredity must be considered, and there are a few rare conditions in which heredity seems to have

been established with reasonable certainty. Amaurotic familial idiocy is one such condition where there is often more than one affected child, although both parents are normal. In a considerable proportion of cases, however, the parents are blood relatives, suggesting that both parents carry the gene for this disorder. While this defect is actually a degenerative process rather than a simple retardation, because the infantile form occurs so early in life it is usually classified with the mental deficiencies.

There are a number of other rare conditions that seem to have a definite genetic basis, some of them due to metabolic disorders, like phenylketonuria where the most striking symptom is severe mental retardation. This condition can be recognized by a very simple urine test, which seems conclusive. A few drops of a 5 per cent ferric chloride solution added to the urine produces a deep blue-green color, which fades in a few minutes. It has been estimated that this disorder occurs in about 1 of every 30,000 members of the population of the United States, but why such a biochemical abnormality should be associated with mental defect is not known.

Another group of conditions in which heredity may at times be a factor consists of the various cerebral palsies. Parental consanguinity with more than one affected child suggests this possibility. Certainly the precise knowledge of human heredity is still so limited that few valid predictions can be made on the basis of established facts, and so far as our knowledge goes at this time, it appears that genetic and environmental causes can produce the same end results.

One of the best known conditions about which there is no general agreement as to causation is mongolism. There is rarely more than 1 mongoloid born in a family and there can be no question that the older mother is far more likely to have such a child than is the younger one. However, in a series of more than 400 such cases in the University of California Medical Center, more than half the mothers were less than 35 years old and many were young and healthy with no signs or symptoms of any physical abnormality. On the other side of the problem is the fact that when mongolism occurs in twins only one of a pair of nonidentical twins is affected, while with identical twins both are affected. This suggests that it is not entirely the mother's physiological condition. In spite of the large numbers of these children (an estimated 1 in every 700 live births), and in spite of the great amount of study and research involved in trying to find a valid cause, there is still no unanimity of opinion as to how mongolism is produced.

Mongolism is one of the few conditions recognizable at birth with a high degree of certainty, not so much because of evidence of mental abnormality but because the facial appearance is very characteristic and the other physical signs are so easily found that a definite diagnosis with a reasonably clear prognosis can be given at once.

Another condition, which may or may not be associated with mental deficiency, has an interesting indirect relationship with heredity. This is the now well-known factor of blood incompatibility between parents both of whom are entirely normal. When the father's blood has the dominant Rh factor and the mother is Rh-negative, there is a great chance that the developing fetus will be Rh-positive and cause a mutual interaction with the mother that may result in mental defect with choreoathetosis in the child. This condition, long known as malignant familial jaundice

producing kernicterus in the child, is now understood so that the mother's blood can be tested during pregnancy for the presence of dangerous antibodies, and preparation can be made to treat the newborn infant before the destructive anemia occurs. It is not unlikely that further research may result in the discovery of other biochemical incompatibilities, which will explain some of the conditions that are now a puzzle to us.

Another disturbance in which early recognition is of the greatest importance is hypothyroidism. This defect is usually not recognizable at birth, but if careful note is taken of the child's general alertness, it may be suspected long before the cretinoid appearance is manifest. A sluggish infant, often with a subnormal temperature, may well be treated at once with thyroid preparations, and if there is recognizable improvement, a tentative diagnosis may be made. Prognosis, however, is not so easy. Prolonged and adequate treatment will usually bring about entirely normal growth and development for a time, but in some cases physical improvement is not matched by mental growth. This may be due to the fact that thyroid deficiency is sometimes but a part of a general deficiency and is not the real cause of the mental retardation. One thing is certain, however, and that is that unless the symptoms are recognized early and treated adequately, the outcome will be poor.

Thus far, only a few striking and readily diagnosed conditions have been mentioned. Most defects do not fall into such groups. It is generally agreed that the great majority of severe defects result from prenatal causes, but there is always some doubt as to whether the prenatal factor is genetic, environmental, or a combination of the two. Obviously, in addition to a careful family history, a study of the prenatal period is of vital importance. It is known that some conditions such as mongolism must have their origin at a very early embryonic age, and there is every reason to believe that adverse factors can occur even before the mother knows that she is pregnant. Although an accurate history of prenatal events is difficult to obtain, every effort should be made to do so. The first two months of pregnancy seem to be most critical for the embryo, and infections that produce a mild or unnoticed illness in the mother may have a serious effect on the child. Such a child often has disturbances of vision with microphthalmia, defective hearing, mental retardation, and sometimes congenital heart disease. When the mother's infection has occurred after the third month of pregnancy, the child does not ordinarily show any abnormalities. If the virus of rubella can cause such ill effects, it is likely that other viruses can behave in the same way, although the evidence for such effects must depend on more complete evaluation of prenatal records correlated with later development in the children.

Certain infections of the mother occurring later during the prenatal period are also known to be transmitted to the child. The best known of these is syphilis, which causes many abortions and stillbirths, but which can also produce a living child who appears healthy for some years only later to develop juvenile paresis. The treatment of a child with congenital syphilis is prolonged and often unsuccessful, but the early recognition of infection in the pregnant mother usually results in effective treatment with a good assurance that the child will not be involved. Other infections have also been demonstrated to have been transmitted from mother to

child. One such condition, toxoplasmosis, may have a mild course in the mother but produce in the child a severe degree of mental abnormality.

Birth injuries also may affect the child, although they are not nearly so common as was previously believed. A prolonged birth with a relatively long period of anoxia may produce abnormality in the child, even if there is no evidence of mechanical brain damage or of cerebral hemorrhage. Prematurity seems to carry special hazards. Certainly the danger of cerebral hemorrhage is greater in the premature infant owing to the fragility of the immature cerebral blood vessels. Of recent years, another accompaniment of prematurity has been noted, apparently related to the administration of oxygen to small, premature infants over a period of time. This is blindness due to retrolental fibroplasia, associated in a number of instances with severe mental retardation and a peculiar, disturbed personality, which seems quite different from that seen in the ordinary blind child or in the infant who has survived prematurity without blindness.

Postnatally there are known cases where infection or brain injury have resulted in mental deficiency in a previously normal child, but these conditions are recognized fairly readily and are far less frequent than are those due to prenatal causes. It is important to remember that some acute brain infections that seem to have been cured may later be followed by definite signs of brain damage, so that the prognosis in any condition of this sort must be guarded.

Until fairly recently, the study and management of mental retardation have not been regarded as within the province of the physician. Today, because of the better management of many of the diseases of childhood, mentally defective persons, who would not have survived a generation ago, may now live into childhood and many years longer. This has resulted in increasing numbers of these children who come to the attention of the practicing physician. Although it is true that medicine usually has little to offer for the cure of mental deficiency, much can be contributed in the way of information that will eventually lead to prevention. Research is now being carried on in genetics and embryology, and the pediatrician, who will increasingly meet cases of mental defect in children, can contribute greatly to the knowledge of causation. There are still many cases for which no cause is known. When there have been complications during pregnancy, it is often difficult to determine whether an abnormal fetus has caused the complications or the complications have produced the abnormal infant.

Since it has been estimated that at least 3 per cent of young children will never be able to manage their own affairs because of mental defect, all knowledge that can be gleaned as to possible causes in so large a problem is urgently needed.

BIBLIOGRAPHY

1. Doll, E. A.: The Vineland Social Maturity Scale. Manual of Directions, Vineland, N. J., The Training School, 1935.
2. Masland, R. L.: The prevention of mental retardation, J. Dis. Child. 95:no. 1, Jan., 1958.
3. Penrose, L. S.: The Biology of Mental Defect, New York, Grune & Stratton, 1949.
4. Stern, C.: Human Genetics, San Francisco, W. H. Freeman & Co., 1949.

Mental Subnormality: Illusions and Directions

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Every physician at one time or another must deal with the problem of mental subnormality. Some cases are diagnosed early, but often the patient is a child in the age range from 5 to 7 years, for it is in these years that children must face school requirements. Since physicians usually want in their diagnostic study an appraisal of the child's intelligence or ability, it is important to give some thought to the problems of appraisal.

Intelligence is not a precise, scientific term, but is an abstract word having a variety of meanings.[7] It is our purpose here to discuss illusions about the meaning of intelligence and management of the subnormal child and to suggest some directions of effort that may lead to a better understanding.

ILLUSIONS

First, there is the illusion that there is agreement on the meaning of intelligence. Some have defined intelligence as the ability that is measured by intelligence tests. Often the intelligence quotient, as obtained from the Stanford-Binet test, is believed to represent intelligence. It is assumed that the ratio obtained by the use of this test is a valid indication of the child's "true" intellectual ability and that the index obtained is all that is required to place the label of "mental retardation" on a child, to place him in a "special class" in the schools, and to cause his parents to join an association of parents of mentally retarded children. Others have sought to define intelligence in biological terms alone. They view intelligence as a physiological characteristic lending itself to laboratory analysis, such as that used to determine blood type. Intelligence is seen as inherited as a unitary trait intact and measurable at any time subsequent to birth.

Modern clinicians view these concepts of intelligence as too narrow and limiting. It is now known that a child can score in the average range on an intelligence test and yet be so ineffective in behavior that he must be placed in an institution. On the other hand, there are persons who do not score in the average range on intelligence tests and yet manage adequately in society. While potentiality resulting from the inheritance of a well-functioning central nervous system is a basis for intellectual ability, learning and experience determine the successful use of that potentiality.[7] In fact, adequate environmental experience is necessary for the expression of the potentiality and its continuing development.

Intelligence can be defined only in the most general terms and a definition must rest on our understanding of the complexity of human behavior. Generally speaking, intelligence is considered to be effective behavior that takes an individual toward goals representing opportunities for fulfillment of needs.[2] Such a definition implies not only the biological factors but also the factors of learning in experience. It

includes the individual's ability to perceive stimuli, his use of experience related to those stimuli, his characteristic problem-solving methods, and his motivation and feeling about his own capabilities.

Much of the misunderstanding about intelligence measurement comes from the fact that those who make intelligence tests are measuring different types of behavior although they all call their instruments intelligence tests.[11] Tests require explicit explanations as to what is to be predicted. It is impossible to speak accurately of "true" intelligence although there are physiological correlates of the type of behavior seen in responses to an intelligence test. In such complex behavior it is impossible to say which physiological variables determine the responses. And further, none can make claim to direct observation of mental processes; it is possible only to make inferences about physiological capacity.

Since the appraisal of intelligence at even a normal level is a complex task, one must recognize how difficult it is to add to the difficulties of appraisal some determination of etiology in dealing with problems of subnormality. The genesis of our problem lies in the complicated structure of the nervous system or in the many possibilities for disorder stemming from factors of the environment influencing development. The sober truth is that we do not know enough about either the biological basis for human behavior or the influence of environmental experience to determine an individual's true ability. Our methods at the moment are makeshift. We need to have more understanding of the structure and functioning of the central nervous system. A child can pass all the tests known to medical science and receive a "clean bill of physical health" and yet be so defective that he is almost unaware of external events. On the other hand, our methods of determining psychological capacities and potentialities are limited and crude. In the past we have paid little attention to creative characteristics of the subnormal child. We know little about his frequent display of limited motivation for problem-solving. We do not know, for example, if this lack of motivation to solve a problem is related to the fact that he is asked repeatedly to perform tasks beyond his capability and thereby comes to see himself as failing before he begins any task.

New techniques and methods of studying the behavior of the mentally subnormal child need to be devised. Clinical psychologists have found projective tests useful in studying personality. These tests are usually comprised of cards with ink blots (Rorschach) or pictures (Thematic Apperception Test) that are indistinct, allowing a variety of interpretations as to meaning. The advantage of these approaches to the study of behavior is that the unstructured stimuli allow a person to put his own meaning and experience into the task of interpretation of the blot or picture.* These tests have not been used extensively in research on the mentally subnormal but they do hold promise.[9] It is probable that lifelike situations would be useful as part of the appraisal process. Clinical psychologists must take into account qualitative factors that often are not made explicit and clear by test scores.[10] It is really life

* At present the writer is studying, with a projective test (a series of pictures such as the Thematic Apperception Test), perceptual and problem-solving characteristics of two groups of mentally subnormal children with mental defect of organic and nonorganic etiologies. These groups are being compared with normal children.[1]

performance that we wish to predict about individuals.* There has been some confusion about such prediction, and some have argued that a standard intelligence test, such as the Stanford-Binet, will predict life performance since it predicts success in school reasonably well. However, academic achievement cannot be a basis for an ultimate determination of mental subnormality unless it is established that scholastic aptitude requires the same thing of a person as does an occupation or a social role.[5]

There has not been enough effort to interpret the behavior of the mentally subnormal person. Many of the concepts and techniques used in interpreting normal behavior can be appropriately used in interpreting behavior of the subnormal. For example, Henry[3] believes that to understand a person's behavior it is important to know about feelings he has had for those close to him in his past and present life. Henry also thinks that the personality takes on consistency in responses to the environment. This seems to be illustrated in subnormal as well as normal children.

Alan, a handsome, neatly dressed boy, 7 years old, consistently lacks courage in meeting new tasks. Whenever he is asked to make a decision or respond to a question, he becomes anxious and upset. Even a very simple problem is difficult for him. He was asked to sit in the red chair in the office and with a frightened expression he hurriedly looked about the room and upon seeing the chair, he exclaimed, "Oh!" Then seating himself, he anxiously looked at the examiner while biting the nail on his right forefinger.

Medical examination shows that he is in good health and somewhat above the normal height and weight. The medical history gives little information that is helpful in discovering the source of his problems, but it is noted that at 6 months of age he had pneumonia with high fever and a year later a high fever and delirium with measles. Residual effects of these episodes cannot be established. His development was slow; he walked at 20 months and said only a few words by the age of 4 years. Thumb-sucking lasted throughout early childhood, and soon after he stopped at age 4, he began biting his nails. Toilet-training was not difficult, but he wets the bed each night. His speech is a problem and the parents see this defect as their greatest concern. Of course, nail-biting, enuresis, and the speech difficulty could all be related to his generalized anxiety.

During Alan's infancy and early childhood, he was left to play alone much of the time as his mother was preoccupied with housework, care of the other children, and problems with relatives. It was only when he entered school and the teacher talked to his parents about his fear and withdrawal that they became concerned about his difficulties. She described him as being quite anxious when given tasks in the normal course of the school day.

Some of the standard psychological tests show that according to the results Alan would be classified as moderately retarded. However, in the tasks of which the test is comprised, he sees threat and becomes so anxious that at times he cannot solve even a very simple problem.

Before Alan can use the ability he does have, he will need much more confidence in himself. A projective test indicated that Alan does not feel loved by his parents and that he sees them as people who are distant and displeased with him. His teacher, although kind to him, is also seen as disapproving of him.

It is apparent that Alan will not be able to meet the school's demands until he has much more confidence in himself. It is necessary for him to have the encouragement any child needs, such as, "We like you, you won't fail, and you can depend on us." Lessening Alan's anxiety will not increase his potential ability, but improvement in his self-confidence will certainly change his performance level.

Although many conditions of an organic nature cause mental subnormality, not

* At the present time, there is in process in the Department of Pediatrics, under the direction of Robert B. Kugel, M.D., a longitudinal research project having as its concern the study of mentally subnormal children of preschool age. This project uses a broad and interdisciplinary approach to study the children in different types of life situations.

all mental subnormality stems from anatomical conditions. There is a type arising from emotional trauma and deprivation. For example, a child may be severely neglected in infancy and lack the opportunity for normal interaction with other human beings. Thus, he will fail to develop normal ability. Kugelmass,[6] among others, holds that extreme deprivation can bring about irreversible conditions of impairment. A child growing up in either urban or rural squalor may be unable to score in the normal range on an intelligence test, nor will his learning experiences bring about skills that will help him to cope with a complex, changing, and demanding society. As our society becomes increasingly complex, persons with inadequate developmental experience may find themselves more and more classified as subnormal. The causes of psychosocial mental subnormality lie deep in the structure of our society beneath the ethical, educational, and economic philosophies as well as realities. One can easily understand the disconcerting aspects of the problem for the practicing physician since means of diagnosis at present are insufficient and many times unreliable. Only the extreme conditions lend themselves to an "open and shut" diagnosis.

A second illusion about mental subnormality is evident in connection with the general usefulness of training and management. In part, the problems stem from a lack of understanding of diagnosis and definition. How can effective and logical training or management be carried out when the causes and conditions are inadequately understood or known? There are many confusing reports of studies showing that under certain conditions and with certain kinds of training, mental subnormality can be alleviated and the problems completely solved. The diagnostic term of mental subnormality has been a wastepaper basket. Sometimes a heterogeneous sample is taken, a training program is applied to the group, and if some change for the better is obtained, generalizations are made. Conditions of children in the sample are often so varied that it is likely some would be benefited by a "shotgun approach." With some children, training, psychotherapy, and even the process of maturation have brought favorable change. But the concept that there is one way of managing or training that is universally useful is an illusion. When parents bring a child and ask "what is wrong?" and "what can be done about it?" the answers available are incomplete and to some extent unsatisfactory.

DIRECTIONS OF EFFORT

What directions should be followed then in dealing with the problem of mental subnormality? First, there should be a recognition that one area of knowledge or one discipline cannot solve the problem of mental subnormality and that there must be contributions from many sources. Social scientists do not have the training and knowledge necessary for understanding the physiological bases of mental subnormality, and physicians have little training or knowledge about research methods and techniques in the social sciences. Each discipline can enhance and extend the usefulness of the other.

An effort should be made to undertake developmental studies beginning during pregnancy and continuing through early childhood in an effort specifically to relate

certain physiological conditions with later behavioral sequelae. A beginning was made in such a direction by Knobloch et al,[5] who studied the effects of prematurity. There may be many conditions during pregnancy or during delivery that are not recognized as having an influence later on behavior. Often physicians and psychologists may try to unravel a developmental history of an older child and find that much of the important information relating to the early life of the child is missing. There must be some way of obtaining and studying valid and reliable data about prenatal, neonatal, and early childhood development and relating this information to behavioral sequelae observed years hence.

Another direction of effort is in the area of improvement in methods of diagnosis. Improvement will be based on more knowledge about human behavior obtained through longitudinal studies of subnormal, normal, and superior children.

Modification of the educational programs for children is needed. Because of inadequate diagnosis and understanding of the many causes of mental subnormality, children are placed in special education classes over the country largely on the basis of the administration of the Stanford-Binet or some other similar psychological test. Since the causes of mental subnormality are multiple, it is unreasonable to expect that the experiences provided in these classes are adequate and beneficial to all children who show symptoms of subnormality. In fact, some children may be impaired in further development by such experiences if their difficulty is of psychogenic origin. As society puts increasing pressure on children to achieve, and as teachers reflect the anxiety in society about achievement, it is likely that the problems of mental inadequacy will increase for the educational system. Much more support and research are needed by school systems for aid in meeting such difficult problems. The philosophy of education in our society about nonachieving persons who are unable to cope with societal pressures toward achievement requires much thought and consideration in relation to over-all societal goals.

The fifth direction of effort should be to place the problem of mental subnormality in a broad setting related to goals that we have for all children. An effort should be made to avoid the necessity of the existence of separate groups of parents; that is, parents of mentally subnormal children in reality want no more for their child than any other parent—they want only an opportunity for their child to have a place somewhere in society. Their goals can be a part of society's efforts for all children. If the needs of mentally retarded children are ignored and society rejects them, parents will seek their welfare without regard for other necessary goals of the community.

A sixth direction of effort should be made toward the betterment of institutions. Institutions need not be dread and distant places. They can be places where society supports an earnest effort to fulfill the needs of individuals and sponsors needed research. Much creative thought and effort can take us far from the forbidding aspect of our institutions of the past as well as many of those in existence today. For many persons, the boundary between institution and community life is too arbitrary—more opportunity for transition and flexibility is needed to make help and facilities available temporarily. Vocational opportunities can be greatly expanded.

PRESENT APPROACHES

The goals and directions just discussed are broad and likely to be of insufficient comfort to the practicing physician at present. There are a number of useful questions that a physician may ask himself in his study of a child. First, has the child mastered his essential developmental motor requirements within the expected time range? Second, does the child use words and combinations of words appropriate for his age and are there spontaneous conversational offerings? Third, does the child respond to objects in a meaningful way? (For example, can he see a toy car as a car or does he see it just as an object to be thrown?) Fourth, does the child depend on the parents for interpretation of objects or social events around him? Fifth, is the child able to bring his own experience and learning into an interpretation of a new situation?

When a diagnosis of mental subnormality is made, the parents need help in dealing with the emotional reactions attendant on the problem. While the parents may often demand specific help and specific answers to many types of questions, these demands may be the reflection of their anxiety and dismay in coping emotionally with the problems. Direct advice often is ineffective. In reality, parents need an opportunity to talk about their feelings and explore them in terms of guilt, inadequacy, and the many deeper aspects of their personalities. At times the family physician can be of help in allowing a parent to discuss feelings and at other times he can refer him to appropriate professional help when necessary. Holt,[4] with much sensitiveness and understanding, explained in a report of a recent study in England the importance of the needs of families with a subnormal child. Of the 201 families interviewed by this physician, many felt that their own physician had given them insufficient time.

Much of what can be done at the present time is palliative, and until our knowledge is greatly increased, our measures will be ineffective and unsatisfactory. Our society may be in danger of lessening some of its humanitarian concern as a result of greater emphasis on scientific advancement, but for it to be a "good" society, it cannot lose sight of the significance and value of individual welfare.

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BIBLIOGRAPHY

1. Alexander, T.: The adult child interaction test, Monogr. Soc. Research Child Development *17*:1-40, 1955.

2. Allen, R. M.: Personality assessment procedures, New York, Harper & Bros., 1958, p. 160.
3. Henry, W. E.: The analysis of fantasy, New York, John Wiley & Sons, Inc., 1956, pp. 7, 17.
4. Holt, K. S.: Home care of retarded children, Pediatrics 22:744–755, 1958.
5. Knobloch, H.; Rider, R.; Harper, P., and Pasamanick, B.: Neuropsychiatric sequelae of prematurity, J.A.M.A. *161*:581–585, June 16, 1955.
6. Kugelmass, I. N.: The management of mental deficiency in children, New York, Grune & Stratton, Inc., 1954, p. 222.
7. McCandless, B.: Environment and intelligence, Am. J. Ment. Deficiency 56:674–691, 1952.
8. McClelland, D. C.; Baldwin, A. L.; Bronfenbrenner, U., and Strodtbeck, F. L.: Talent and society, Princeton, N. J., D. Van Nostrand Co., Inc., 1958, p. 9.
9. Molish, H. B.: Contributions of projective tests to problems of psychological diagnosis in mental deficiency, Am. J. Ment. Deficiency 63:282–293, 1958.
10. Sarason, S. B., and Gladwin, T.: Psychological and cultural problems in mental subnormality: a review of research, Genet. Psychol. Monogr. 57:3–290, 1958.
11. Spiker, C. C., and McCandless, B. R.: The concept of intelligence and the philosophy of science, Psychol. Rev. *61*:255–266, 1954.

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PHARYNGITIS THERAPY. A new throat lozenge containing a synthetic quaternary-ammonium type of antiseptic plus benzocaine (Bradosol, Ciba) relieves pain and destroys most of the organisms associated superficially with sore throat. The drug is reported effective against fungal growth as well as against virtually all gram-positive and gram-negative bacteria, including antibiotic-resistant strains of Staphylococcus aureus tested in vitro.

VISUALIZATION OF GALL BLADDER. A new oral diagnostic agent that permits optimal gall bladder visualization with just one dose, regardless of the patient's weight, was introduced by E. Fougera & Co., Inc., Hicksville, N. Y., at the meeting of the American Roentgen Ray Society. Called Orabilex, this cholecystographic medium radically reduces the incidence of side effects to less than 5 per cent. The standard dose is 6 capsules to be taken approximately 12 to 14 hours before the examination.

PREMATURE INFANTS. A form of milk with double the caloric content of usual premature baby formulas promoted weight gain and growth rate greater than that usually seen in premature infants fed breast milk or ordinary formulas, reported Dr. Hans G. Keitel, Philadelphia, at the American Medical Association clinical meeting in Minneapolis. Infants regained their birth weight more rapidly, could sometimes be discharged days or a week earlier.

DRUG RELIEVES SYMPTOMS OF URETERAL COLIC. The symptoms of ureteral colic usually caused by the passing of small stones or gravel from the kidneys have been relieved in 47 of 50 patients treated with a new form of oral atropine (oral atropine tannate, Atratan, Irwin, Neisler & Co.). Of 40 patients suffering from ureterolithiasis, 39 were benefited by the drug (1 mg. tablets); it was "of distinct value" in 8 patients with ureteral colic due to causes other than stone. These findings were reported at the A. M. A. clinical meeting.

NEW CERVICAL SPOON. An improved cervical spoon for sperm collection consists of a concave plastic lining for the posterior vaginal wall (Obst. & Gynec. 12:433, 1958). It gently curves over the perineum and permits coitus without

discomfort; its use can easily be demonstrated in the office. The seminal pool pH (7.0 to 7.5) has been preserved for eight hours in vivo with the use of this spoon.

CANCER BLOOD TEST. In a new cancer blood test, homogenates from cancer tissues, together with Freund's adjuvants, are subcutaneously injected in female guinea pigs; when sacrificed their uterine horns are immersed in a Schultz-Dale bath. The addition of the proper antigen (contained in serum from cancer patients) produced a local anaphylactic response owing to the antigen-antibody union. A positive result was obtained in 96.8 per cent of carcinoma cases. The technique was discovered by Dr. Jack G. Makari (Plainfield, N. J.).

PERIPHERAL VASODILATOR. Recently introduced was cyclandelate (Cyclospasmol, Ives-Cameron), indicated in vasospastic and obliterative disorders such as intermittent claudication, leg ulcer, and Raynaud's disease. Extensive clinical studies of the drug in Europe have shown that increased blood flow occurs within 30 minutes. Severe side effects have not been observed, nor have cases of hypotension.

PATIENT-CYCLED RESPIRATOR FOR EMPHYSEMA. An iron lung that keeps time with the patient's breathing is being used for seriously ill emphysema patients by Dr. John Mason Tyler at Lemuel Shattuck Hospital, Boston. The patient-cycled respirator is designed to respond to a signal from a gauge placed around the patient's chest. The apparatus is then regulated to function with the normal respiratory cycle. The respirator can be adjusted to both the timing and depth of the patient's breathing.

NEONATAL CIRCULATION MECHANISMS. A fundamental change in neonatal circulation mechanisms may occur at 4 to 5 days of age, it was indicated in a study of 71 normal newborn infants during the first 12 days of life (Brit. M. J. 2:276, 1958). Systolic blood pressure was usually unaltered during crying or tilting to the feet-down position in the first 3 to 5 days, whereas during the next week, both crying and tilting raised systolic pressure.

ANTIPRURITIC. A new topical corticosteroid, triamcinolone acetonide (Kenalog cream, lotion, or ointment, Squibb), is indicated for many inflammatory skin conditions, including atopic, contact, and eczematous dermatitis, neurodermatitis, and seborrheic dermatitis. In addition to prolonged relief of itching, the drug is also said to exert marked anti-inflammatory and antiallergic action. Systemic toxicity has not been observed.

Victor Robinson: A Romantic Medical Historian*

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Early in 1946, a Committee of Sixty was formed to sponsor the publication, in honor of his sixtieth birthday, of what was to have been called the *Victor Robinson Anniversary Volume*. But the Fates willed it otherwise, and the volume intended to honor Dr. Victor Robinson commemorated instead the first anniversary of his death. For, on Jan. 8, 1947, "at ten o'clock in the morning, while sitting in his library in his large, comfortable chair, reading *William Henry Welch and the Heroic Age of American Medicine*, the book slipped from his hands, and in that moment, he was gone."[1]

The *Memorial Volume* appeared in 1948 as a tribute and a testimony of esteem from colleagues, former students, and friends of Dr. Robinson. By their very nature, however, *Festschriften* and similar appreciative publications are limited in their ability to renew continually and to transmit into the future the contributions, attributes, and qualities for which a man has been valued and honored by his contemporaries. And only too often, as those who knew him are themselves gathered to their fathers, memory pales and leaves for future generations but a name, a book or two, and perhaps a shadowy figure based on apocryphal anecdotes. For this reason it is deeply gratifying that we are gathered here today to inaugurate a memorial to Victor Robinson, which can keep alive not alone his name, but even more significantly the distinctive qualities, interests, and values that characterized him as a man, as a scholar and author, and above all, as a teacher.

When I was honored with the invitation to give the First Annual Victor Robinson Lecture in History of Medicine, it seemed most fitting and appropriate to talk about Victor Robinson, the medical historian. Two opinions motivated this choice. In the first place, despite the recognition he received, I have long felt that his contribution to the history of medicine has been underrated and that he has not been given all the credit he deserves. Second, an analysis and assessment of Robinson's approach to the history of medicine, his aims as a historian, and the means employed to achieve them would provide a firmer basis for an evaluation of his achievement.

Any endeavor to assess the distinctive contribution of the historian Victor Robinson must take account of the man. All who have written about him have remarked on his personality. Modesty, gentleness, a keen sense of humor, and an ability to listen were qualities that endeared him to his friends. No one who heard him lecture, or who met and talked with him, ever forgot him. He had a sensitive appreciation of and a vital interest in people, coupled with an understanding of the vagaries and peculiarities of human behavior. The grain of the man may be glimpsed from an autobiographical sketch written not long before his death.

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* First Annual Victor Robinson Lecture in History of Medicine, Temple University School of Medicine, Sept. 23, 1958. Published through the kind courtesy of Dr. Fred Rogers.

> There is nothing in my career to excite a publicity department [he wrote]. Father was a prosperous physician, and the author was brought up in a bourgeois household of comfort and culture. I never walked the streets looking for a job (never had a job of any kind); never was hungry (except between meals) and have never been arrested (as yet). So you see, I do not make "good copy."
>
> Mary Lou McDonough tried to make an interesting story of my life when she included three of my poems in her anthology of medical poetry written by physicians.* Mrs. McDonough submitted a biographical sketch in which she tried to make much of the fact that I had accomplished so much although coming from the little Ukraine. I had to scotch the story, because the Ukraine is not so little, and because I arrived in this country in my infancy—therefore my birth in the Ukraine had nothing to do with the case.
>
> As I had the advantage of education, I cannot consider myself a self-made man. In one respect, however, I am self-taught: The History of Medicine and Allied Sciences. There were no courses in these subjects in my student days, and I may be considered the first American "specialist" in the History of Medicine, as I was among the earliest to devote almost full time to this specialty. I founded and edited *Medical Life* (1920–1938), the only monthly of medical history in the English language. In 1929, I became Philadelphia's first professor of the History of Medicine when this chair was created for me at Temple University School of Medicine.[2]

This modest, even self-deprecatory sketch leaves much unsaid. But, while the "frontage," to use Browning's term, does not fall, there are hints in this outline to some of the factors that formed Victor Robinson, the man and historian. While he came from a middle-class family, the household in which he grew up was not staid and conventional, as his description of it seems to imply. Actually, it was a lively, colorful household, where a sensitive, receptive young man was exposed to provocative currents of thought and unconventional, even iconoclastic ideas.

His father, Dr. William J. Robinson, was an outspoken proponent of a sane outlook in matters of sex and one of the most indefatigable workers for contraception. In 1904, he began a vigorous educational campaign to make available information on the subject and to drive home the need for a sensible, reasonable attitude. One of his major targets was legal interference with the right of the physician to provide contraceptive advice when he felt it medically and socially indicated. William Robinson addressed himself to the medical profession and the general public through his numerous publications. A prolific writer, he drove home his ideas through numerous popular books on sex, as well as in professional publications, especially his periodicals, the *Critic and Guide* and the *American Journal of Urology*. In the opinion of one historian, "Few American physicians of our time . . . have had more influence in moulding opinion on contraception than Robinson."[3]

Equally influential in molding Victor Robinson was his mother. When he published his essay on William Godwin and Mary Wollstonecraft in 1907, he dedicated it to her "with the hope that in coming years it will bear fruit which will in some measure realize the dreams she dreamt when she rocked her first cradle and sang her first loving lullaby."[4] That the sentiments and ideas expressed in this essay were in part, at least, an outgrowth of these "dreams" appears very likely. The beliefs that human existence could be improved, that man could be led toward a better future, that man was deformed by evil institutions, that liberty and freedom were the highest values,

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* "The Healing Hands," "Tuberculosis," "Hasheesh." *In:* Poet Physicians, Springfield, Ill., Charles C Thomas, 1945.

and that these aims could be achieved through the agency of rational knowledge—these were among the ideas with which Victor Robinson grew up and which he observed.

In his first publication, he took his stand boldly.

> . . . if what Godwin fought for and Mary Wollstonecraft worked for is a dream [he wrote], it is a dream to which I must cling. . . . It is the dream that some day the human race will exist without a Government upheld by the bayonet of the soldier and the club of the policeman; that our jails will be empty of prisoners and our streets of beggars; that the whirr of cotton mills will not drown the sobs of babies; that breaker boys and old men will not sit in the coal mines year after year and wear their lives away; that women will not be compelled to toil in prison factories five hours before their children are born, and thus give birth to poisoned offspring. It is the dream that some day all will have enuf [*sic!*] bread to eat, enuf clothes to wear, a home to lay their heads and just a little time to love, loaf and laugh; the dream that the serpent with a hundred fangs, the Medusa with a thousand faces, the leech with a million mouths, will loosen their terrible grip from the toiler's bleeding throat; that labor will receive what labor earns; that they who create wealth will not be penniless; that they who feed us will not starve; that they who clothe us will not wear rags; that they who house us will not dwell in hovels; the dream that Chauvinism . . . will disappear, and phoenix-like, from the ashes will be born the desire to be the Universal Brother; the fond dream, the sweetest hope of all, that war, the worst of crimes, will cease, and the soldier, the worst of slaughterers, the bravo butcher—the useless wholesale murder—will no longer flourish; the dream that some day all the nations of the earth, black and white, red and yellow, will be links in the grand unbroken chain of mutual friendship.[4]

The great obstacle that blocks the realization of this goal is authority. Victor Robinson, at the age of 21, saw authority as "the dam which has blocked the River of Progress. It is the clog in the wheel of improvement, the barnacle on the ship of Science, the dark cloud which obscures the dawn of day, the radiance of reason."[4] And he believed just as ardently that in the struggle of mankind for freedom and progress, the advances were made by pioneers and pathfinders, by men and women with the courage to be innovators, to oppose evil and error, to reject accepted ideas, and to stand firm in the cause of truth. Scientists, philosophers, freethinkers, fighters for individual freedom and social justice were the vanguard of progress.

In line with this view of history and social development was the publication by Robinson in 1908 of an essay on Peter Kropotkin, the Russian price who became a philosophical anarchist. This was the second in a series entitled "Lives of Great Altrurians." Subsequent publications were to be devoted to Robert Ingersoll, Walt Whitman, Giuseppe Garibaldi, Thomas Paine, Élisée Reclus, Louise Michel, Grant Allen, Elizabeth Cady Stanton, Alexander Herzen, Ferdinand Lassalle, Charles Darwin, and Ernst Haeckel. The title of the series apparently derives from *The Traveller from Altruria* (1894), the Utopian romance by William Dean Howells in which the ways of capitalism were subjected to critical analysis. Thirteen years later, in 1907, Howells completed his Utopian account with *Through the Eye of the Needle* where he suggested the more rational society that might be possible on the basis of cooperation and altruism. At any rate, none of the projected essays were published, except one on Darwin, which appeared originally in 1909 in the *Medical Critic and Guide* and was then reprinted in the first edition of *Pathfinders of Medicine*.

A further light on this period of Victor Robinson's life was cast by his sister,

Ellen H. Ringer. "In 1910," she related "he made the first of many trips to Europe. After visiting all the leading cities, he made a pilgrimage to the rarely-visited Caprera, the wild island home of Garibaldi—the Italian Liberator. Garibaldi's widow gave him some flowers which she plucked from the hero's tomb, and which were carefully preserved through all the years."[5]

Victor Robinson grew up in an environment when optimism seemed justified, when hope for the future seemed a natural and logical extension of past history. His mind was formed at a time when it was possible to believe that poverty, disease, war, and other social evils would continually diminish through social reconstruction, with rational knowledge and enlightenment as the chief agents in this process. It was a time of great hopes, a period that enjoyed an intoxicating sense of freedom, a desire to be free of anything and everything that smacked of constraint. Politically, it was the era of the new freedom, but it was also the period of the new history, the new poetry, the new woman, and it was also the first great period in the creation of American social policy and legislation in relation to health and welfare. It was, to use a label, a period of social romanticism, and as is not uncommon in such periods, it had its exaggerations and eccentricities.[6] These tendencies were in varying degree reflected in the attitudes, the interests, the sympathies and antipathies of Victor Robinson when he first entered upon his career as a writer and historian.

It took several years, however, before he found his true vocation, the study and teaching of medical history. In casting around for a profession, he studied first at the New York University Law School, but finding the law arid and uncongenial, he turned to pharmaceutical chemistry, receiving a Ph.G. degree from the New York College of Pharmacy in 1910 and a Ph.C. degree from Columbia University in 1911. But pharmacy could not satisfy him, and he went on to study medicine, obtaining his M.D. degree in 1917 from the Chicago College of Medicine and Surgery (now the Loyola University Medical School).

The 10 years from 1907 to 1917 were a transitional period in the life of Victor Robinson. He was seeking a field of activity that was congenial with his interests and in which he might fruitfully apply his talents. The year 1912 marks the turning point in his development. He published a volume of poems, an *Essay on Hasheesh*, and the first edition of *Pathfinders in Medicine*. Early in 1912 Robinson had become a contributing editor to the *Medical Review of Reviews*, in charge of its Department of the History of Medicine, and most of the essays in *Pathfinders* were originally prepared for this publication. In the same year he also founded the Froben Press to make available for those "who are working in the cultural fields of medicine" publications for which a wide distribution could not be expected. The events of 1912 point to the road on which he would find his true calling, the road to Clio Medica. Having become fascinated by the pathfinders, the innovators, the rebels in medicine, by "those who enlarge our mental vision" in overthrowing the established dogmas, he turned to the history of medicine as a field congenial to his impulses and on which his talents might be brought to bear. With the completion of his medical studies, Victor Robinson was ready to become the first medical historian in the United States to devote his entire time to this subject.

Here two questions are in order. What was the condition of medical history in

this country when Robinson came upon the scene? How was he equipped for the task he had set himself? Answers to these questions provide the basis on which an evaluation of Robinson the historian can be developed.

While some evidence of interest in the history of medicine can be found in the United States during the early nineteenth century, the first large impetus toward the furtherance of the subject came from John Shaw Billings through his lectures at the Johns Hopkins University.[8] Having been appointed Lecturer in the History of Medicine in 1876, he gave a course of 20 lectures on medical history, medical legislation, and medical education a year later. In 1891, while holding the post of Lecturer in Municipal Hygiene, Billings gave another series of lectures on the history of medicine. With the opening of the Johns Hopkins Medical School in 1893, he received an appointment as Lecturer in the History and Literature of Medicine, a position he held until 1905 when he resigned. It is not surprising, therefore, to find Baltimore playing a distinguished part in the medical history movement in the United States. The founding of the Johns Hopkins Hospital Historical Club by Osler, Welch, Kelly, and others in 1890 acted as a further stimulus in arousing interest.

There is evidence that active interest in medical history was becoming widespread around the turn of the century. Roswell Park began to teach medical history at the University of Buffalo in 1897, and in 1901 the following were also mentioned as teachers of the subject: C. W. Dulles in Philadelphia, Thomas Gray at Minneapolis, C. L. Nichols in Boston, Burnside Foster in St. Paul, and Sarah Hackett Stevenson at the Women's Medical School in Chicago. It is quite likely that sporadic instuction was given in other medical institutions, usually by some member of the faculty interested in the subject. Alfred E. Cohn remarked that when he went to medical school, at the turn of the century, the professor of physiology "gave a few lectures on the history of the discovery of the circulation of the blood."[9]

The survey that Eugene F. Cordell made in 1904 probably gives the best picture of the place of medical history in the American medical school in the first decade of the century. He investigated conditions in 14 leading medical schools and found that only three were giving full courses of 14 to 16 lectures. As for the others,

> there are four "lectureships," one just established and still without an incumbent, and another held jointly with a "Clinical Professorship of Dermatology." One of the "courses" consists of three lectures! There is but one professorship, and that an "honorary" one. In two institutions "some" instruction is given by the Professor of Therapeutics and the Assistants of Surgery respectively; in the latter case only in surgery. In one, and that one, strange to say, Harvard, lectures were attempted, but "no great interest" was shown and they were discontinued. There is no uniformity in those receiving instruction; sometimes it is the sophomores, sometimes the juniors or seniors, and sometimes any that *choose* to attend. In but one is the claim made that the course is compulsory. In none is there any examination. One can readily imagine what the attendance must be under such circumstances, and the experience of Harvard is instructive.[10]

Ten years later, Arnold Klebs again looked at the place of history in the medical school and concluded "that the study of the history of medicine has [not] made very decided progress as a feature in medical education."[11] He did point out, however, that the number of academic courses on the subject was not an exact measure of the interest manifested in the subject itself. And, indeed, there were indications of

activity and interest in medical history. Several journals concerned with medical history made their appearance. The *Medical Library and Historical Journal*, later the *Aesculapian*, was published from 1903 to 1909. The Chicago Society of Medical History was organized in 1911 and began to issue a *Bulletin*. In 1917 the *Annals of Medical History* made an entry on the scene under the aegis of Paul B. Hoeber and Francis R. Packard. The first edition of Garrison's *History of Medicine* was published in 1913. Several physicians emphasized the value of having the medical man study the evolution of his field of endeavor. In 1916, Robert E. Schlueter insisted that medical history is necessary for "the completion of a general education, for the fixation of technical knowledge, the prevention of professional narrowness and the ennobling of character."[12]

The situation in the United States at this time was part of a movement in both Europe and this country that at the end of the nineteenth century and during the first two decades of the twentieth century brought about a renaissance of medical history. It was within this movement that Victor Robinson embarked on his career and made his contributions as writer, as editor and publisher, and as teacher.

For these roles he was qualified both by talent and by circumstance. At heart he was an artist, more interested in poetry than anatomy, but he had the rare ability to see the poetry of science. Moreover, he was able through the skilled use of language to transmit to others and to arouse in them what he felt, what he imagined and thought. Robinson painted with words, fully aware of the effects that could be produced, and consciously developing his medium by cultivating a style. This was already evident in his *Essay on Hasheesh* where he described his sensations after taking 20 minims of *Cannabis indica*.

> I hear music [he wrote]. There is something strange about this music. . . . In the soft surge and swell of the minor notes there breathes a harmony that ravishes the sense of sound. A resonant organ, with a stop of sapphire and a diapason of opal, diffuses endless octaves from star to star. All the moonbeams form strings to vibrate the perfect pitch, and this entrancing unison is poured into my enchanted ears. . . . I float into the outside air. Sweeter and sweeter grows the music. . . . and I float in tune with the infinite—under the turquoise heavens where globules of mercury are glittering. . . . I turn, I twist, I twirl. I melt, I fade, I dissolve . . . .I am on the top of a high mountain-peak. I am alone—only the romantic night envelops me. From a distant valley I hear the gentle tinkling of cow-bells. I float downwards, and find immense fields in which peacocks' tails are growing. They wave slowly, to better exhibit their dazzling ocelli, and I revel in the gorgeous colors.[13]

This quality is to be found in his historical writing, adapted naturally to the subject, and it was this talent to paint with words that made Victor Robinson also an effective teacher.

The ability to create history as literature was paralleled by a capacity for recreating vanished people and events. His approach to medical history was in terms of individuals with whom he established an empathic relationship. He lived, suffered and rejoiced, wept and laughed with his heroes. His approach is perhaps nowhere better exemplified than in his review of the memoirs of Linda Richards.

> The chief defect of Linda Richards' autobiography is its absence of human note [he said]. This seems an impossible charge to be brought against one where entire life was devoted to

> nursing and in training others to nurse. But nowhere in the pages before us can we find any outburst of feeling. Between the covers of this book there is no gleam of humor, no hint of romance, no little weakness over which to smile indulgently, while anything approaching an indiscretion is altogether unthinkable.[14]

Just the opposite was true of Victor Robinson. Perhaps the best description of his idea of the task of the historian was written by a man whom he respected but whom he never met. Mortimer Frank, in reviewing the second edition of Garrison's *History*, wrote:

> We ask of the historian, not to load our memory with facts but to recall the dead past to life, to give us a vivid, animated, and truthful picture of the times that are no more. We require him to make us live the life of our medical forefathers; to initiate us into their ideas, their beliefs, their passions; to disclose to us all the motives good or evil, on which they acted; to reveal to us their virtues and vices; and we thus say of the historian, what used to be said only of the poet, that he must be a painter.[15]

But Robinson the poet, the man of creative imagination was also endowed with boundless energy. While convalescing from his first heart attack in November, 1946, he described himself as one . . .

> Who had never been sick, who knew no pain except a toothache;
> Who sat up late working under tension, chain-smoking incessant cigarettes;
> Who went to Katz's for hot-pastrami sandwiches, french fried potatoes, pickles and beer;
> Who was as strong as a horse, and shoved heavy desks around for exercise;
> Who had no time to spare for a vacation;
> Who kept going at sixty as if I were still a youngster. . . .[16]

This energy went into the creation of channels of communication for all those interested in the history of medicine. Most important of these was *Medical Life*, which was founded in 1920 and continued to appear until 1938. It was published initially in July, 1920, as an eight-page *Medico-Historical Bulletin*.

> Letter writing is an art that was lost in the nineteenth century [Robinson wrote], but aside from the mail there exists no agency by which our workers in medical history can keep in touch with one another. . . , the Medico-Historical Bulletin will endeavor to be the medium by which the disciples of Toner and Handerson can exchange wants and interchange ideas; it will report the news in this field, and will review the latest contributions to the subject.[17]

In October, the *Bulletin* was transformed into *Medical Life* and became the first monthly journal in English to be devoted to the history of medicine. Robinson opened the pages of *Medical Life* to eminent historians and to beginners alike. A number of outstanding European medical historians contributed papers and served on its editorial board. Among them were Max Neuburger, Karl Sudhoff, Henry E. Sigerist, Arturo Castiglioni, John D. Comrie, and Wilhelm Haberling. Robinson met a number of European historians in 1922 when he attended the International Congress of the History of Medicine. Henry Sigerist in 1946 recalled his first meeting with him.

> When I look back, I remember the summer of 1922 when we first met in London . . . .We were all young at that time. You had come from America to make contacts in Europe. I was an unknown lecturer in the history of medicine of the University of Zurich. All I had to my

credit were a few papers and the fact that I was a student of Sudhoff and had a few books in the press. But the books were not out yet and I was at best "a promising young man." You were only a few years older than I but you had started writing books at the age of twenty and you were editor of a monthly, the only monthly journal devoted to the history of medicine.

I felt greatly honored when you invited me to join the Editorial Board of *Medical Life* with such men as Sudhoff, Neuburger, Singer, Castiglioni. You had several of my papers translated into English and it was you, and no one else, who introduced me to the American public.[18]

Medical Life also became the official organ of the American Society for Medical History. But while making it possible for American and European students of medical history to publish their work, Victor Robinson continued his own literary activity. *Pioneers of Birth Control* and *The Don Quixote of Psychiatry* appeared in 1919, followed by *The Life of Jacob Henle* in 1921. There were no other books from his pen until the end of the twenties, but he prepared numerous papers, for the most part biographical in character. Most of the biographical papers were eventually included in the second edition of *Pathfinders in Medicine*, published in 1929. Occasionally, there were articles on nonbiographical subjects, but these were relatively rare in this period. At the same time, Robinson was changing and developing, both philosophically and professionally. Of this process the written record offers occasional but fascinating glimpses. As one might expect, the decade of the twenties, the atmosphere of "normalcy," of Babbittry was not congenial to Victor Robinson, and in the preface to the second edition of *Pathfinders*, he gave voice to his disillusionment and the hope to which he still adhered.

During that spiritual journey in which we all partake [he wrote], and the more perilous the voyage, the more imperative that we embark—the author entered many temples and knelt before many shrines; the idols of his early days have fallen from their pedestals, and the Truth lay reachless beyond the abandoned altars. . . . We live in the renaissance of machinery, but in the decadence of the human spirit. Across the deepening twilight of the dying faiths we seek an answer on a new horizon, and the only reply is the million-voiced roar of the Motor. Even before our era, John Addington Symonds asked, "What is left for us modern men? We cannot be Greek now," and he answered, "Hasheesh, I think: Hasheesh of one form or another." The author's particular form of Hasheesh has been medical history. Interest in the History of Medicine has persisted, for it is the greatest of stories—the record of man's conflict with credulity.[19]

It is characteristic of Victor Robinson that he did not lapse into cynicism. There was still room for hope, but hope was no longer the wild, unfettered creature certain that all was possible. Now there was a profound awareness of the limitations and obstacles that made difficult the human condition and the human quest. This philosophy of life can hardly be better presented than in his own words. In *The Story of Medicine*, published in 1931, Victor Robinson wrote:

Life is a great experiment in physiology, in which we all take part: the unknown holds the protocols, and Death writes the conclusions. We do not know where the experiment is tending, nor can we control the outcome. There are gates we cannot open, and impossible roads without a guide-post. Yet we have pressed immeasurably forward; the sable curtain still shrouds us in darkness, but it is no longer so frightening. Often it is lifted, and where once was most impenetrable, are seams through which we steal a glance at the mysteries beyond. We have mounted our present height over the accumulated discoveries of the centuries, but chiefly those of modern times. We linger still on the lower wings, and the upward climb is

> uncertain, but never was the way so clear. In the eternal quest, Medicine and Biology are halted by a speck of protoplasm, within whose cell is locked the mystery of animated matter. With ever new, yet ever insufficient knowledge, we continue the never-to-be-ended Journey.[20]

This was the philosophy behind his activities of the thirties and forties. His writings of this period, *The Story of Medicine*, *Victory Over Pain*, and *White Caps*, are all imbued with this *Weltanschauung*. And it was with this philosophy that he entered on his career as a teacher, for in 1929 the Temple University School of Medicine created for him a professorship of medical history. To this new task he brought not only a mature view of life but also a more broadly based technical equipment. When Victor Robinson first decided to become a medical historian, he possessed certain great advantages: enthusiasm, love of books and literature, and a technical knowledge of pharmacy and medicine. On the other hand, his linguistic equipment and his training in the uncovering of historical data were limited. By the end of the twenties these defects had largely been remedied. He had always been an omnivorous reader with broad interests, and reading continued to be an important tool for him. Along the way, he also picked up a good working knowledge of various European languages. Equipped with an excellent memory, wide knowledge, creative imagination, an interest in and understanding of people, and an ability to talk well, Victor Robinson was a teacher able to pass the spark of enthusiasm and to kindle the fires of interest in students.

His approach to the teaching of medical history and the scope of his course were presented in 1930 in *Medical Life*.

> The course in the History of Medicine is a pictorial one, being illustrated with about five hundred slides. This graphic presentation enables the student to see the history of his profession as a developing panorama throughout the ages. The following subjects are emphasized and illustrated:
>
> I. Primitive Medicine—origin of trephination, bloodletting and therapeutics.
> II. Egyptian and other ancient forms of medicine.
> III. Greek Medicine, including Alexandrian and Roman.
> IV. Medieval Medicine—cessation of the Experimental Method.
> V. Medicine in the Renaissance—Reawakening of the Experimental Method.
> VI. The Quest for Human Anatomical Material for Dissection.
> VII. Anatomy and Physiology in the Seventeenth Century.
> VIII. Medical Leaders and Chemists of the Eighteenth Century.
> IX. Pathfinders in Medicine in the Nineteenth Century.
> X. Landmarks in American Medicine.
>
> In unravelling the development of any science, we unwind the first uncertain strands in Egypt and Babylon, proceed properly in Greece, slacken our pace among the Byzantines, lose the thread in the twilight of medievalism, then pick up the long-broken skein in the Renaissance and carry it forward to the present day.[21]

There is no doubt that the course was well received. The use of visual aids and the skillful employment of anecdotes made it possible to create a receptivity for more serious aspects of the subject. Furthermore, the sincerity and enthusiasm of the teacher impressed the students during the 18 years in which he held the professorship. During this time Robinson had to get up early in the morning to make the seven o'clock train to Philadelphia so as to reach the classroom in time for his nine o'clock

lecture. The position was unsalaried, and it is clear that this was a work of love. The impression that he left may be measured at least in part by the fact that we are gathered today to perpetuate his memory and his work.

This then was the career of Victor Robinson, medical historian. We have looked at some of the elements that entered into its growth and development, and the questions may now be raised: What was his contribution? Where does he stand in the field of medical history? What in his work and thought must we preserve? In part, the answers have already emerged. Victor Robinson was a member of the generation that created medical history in this country as we know it today. He banged the drums for medical history, he blew its horn, he was a propagandist for medical history. He aroused interest in the subject and made it possible for interested persons to participate in the medical history movement. He accomplished this through the channels of communication that he created, chiefly *Medical Life*. He was also a transmission belt by which currents and personalities of medical history in Europe were introduced to Americans.

To be sure, he was not alone in this endeavor. One cannot overlook other men concerned with medical history, such as Osler, Welch, Kelly, and Streeter, who stimulated interest in the history of medicine through their personal enthusiasm. But there was a difference. Henry Viets has characterized the members of this group as "men of scholarship, charm, book collecting instincts and the long purse."[22] For them the history of medicine, even when taken seriously, was still secondary to other interests. For Robinson it was the center of his professional life, for he was a professional historian. He was not a collector of rare books per se; he was not a bibliophile in the collector's sense. He loved books, but he was more interested in the men who wrote them, how they came to write them, and what they meant. This endeavor to feel his way into the lives and thoughts of people who lived hundreds of years ago is a marked characteristic of Victor Robinson, from which one can still profit. An insight into the thoughts, emotions, reactions, likes, and dislikes of people of the past requires a projection of ourselves into the past. In this way we can learn something of ourselves and develop a sense of perspective with regard to our own situation.

Medical history in this sense is a tool that enables one to acquire a degree of immunity against uncritical enthusiasms, against faddism in medicine, while helping one to recognize the ever-expanding and widening frontiers of knowledge. Victor Robinson held fast to this viewpoint. While he recognized the weaknesses and frailties of human beings, he retained an unswerving belief in the importance of seeking the truth and of the utility of reason in achieving this goal. Moreover, although he left behind his early enthusiasms for social panaceas, he retained to the end a sensitive social consciousness. He sympathized with the underdog, and he did not hesitate to call attention to social injustice. His last book, *White Caps* (1946), is not only a history of nursing but also a plea on behalf of women as an underprivileged group.

These are some of the meanings of medical history as Victor Robinson understood it, wrote it, and taught it. There is, however, another element in the testament of Victor Robinson, which underlies all I have said and which is perhaps most important.

In this connection a personal note will, perhaps, not be out of order. In 1938, I became the editor of *Ciba Symposia*, a journal of cultural and historical medicine, which was also the organ of Ciba Pharmaceutical Products, Inc. It was my job to organize this publication, which at that point was only a project on paper. One of my tasks was to secure competent authors, and not unnaturally I turned to Victor Robinson. He became a contributor and wrote on trepanation, aviation medicine, hashish, and other subjects. Not only did he contribute manuscripts, but from his vast fund of knowledge made useful suggestions, provided illustrations, and helped to make the particular issue with which he was involved a success. Then, when a group of New Yorkers interested in medical history founded a society in 1940, Victor Robinson quite naturally became the first president. I was a charter member of this group. As a result of these circumstances, it was my privilege to visit him on a variety of occasions and to have numerous opportunities for unhurried conversations. Among my most cherished recollections are those of various afternoon visits at Four St. Luke's Place. Outside, the subdued rumble of traffic, the shouts of children at play, and all the other sounds that make up the tonal environment of downtown New York. But seated at Robinson's desk in his library, one heard little of all this. We conversed, spoke about the eternal problems of working with printers, with authors—I was then a very young editor—about current events, our favorite figures and periods in the history of medicine, about the societies concerned with medical history. At some point in the conversation he would push his glasses high on his forehead and look out the window, peering into the distance as if seeking the answer to a problem. This characteristic pose epitomizes the most important value in the testament of Victor Robinson, whose whole life was dedicated to the search for truth, to the unending search for truth so that each generation may in some small measure diminish its ignorance and advance its knowledge in order to improve the lot of mankind. And in this quest, Victor Robinson believed that medicine and its story could play an important part.

BIBLIOGRAPHY

1. Kagan, S. R., editor: Victor Robinson Memorial Volume. Essays on History of Medicine in Honor of Victor Robinson on His Sixtieth Birthday August 16, 1946, New York, Froben Press, Inc., 1948, p. ix.
2. Rosen, G.: Victor Robinson (1886–1947), J. Hist. Med. 2:117–120, 1947.
3. Hinds, N. E.: Medical History of Contraception, Baltimore, Williams & Wilkins Co., 1936, pp. 310–311.
4. Robinson, V.: William Godwin and Mary Wollstonecraft. (Lives of Great Altrurians), New York, The Altrurians, 1907, pp. 18, 19, 21–22.
5. Ringer, E. H.: Victor Robinson, Ph.C., M.D. *In*: Kagan,[1] p. 444.
6. Goldman, E.: Rendezvous with Destiny, New York, Alfred A. Knopf, 1953, pp. 223–225.
7. Rosen, G.: A History of Public Health, New York, MD Publications, Inc., 1958, pp. 463 ff.
8. Rosen, G.: The place of history in medical education, Bull. Hist. Med. 22:594–627, 1948 (especially pp. 605–606, 613–617).
9. Cohn, A. E.: No Retreat From Reason, New York, Harcourt, Brace & Co., 1948, p. 252.
10. Cordell, E. F.: The importance of the study of the history of medicine, Med. Library & Historical J. 2:268–282, 1904 (p. 270).
11. Klebs, A. C.: The history of medicine as a subject of teaching and research, Bull. Johns Hopkins Hosp. 25:1–10, 1914.
12. Schlueter, R. E.: The necessity for studying medical history, J. Missouri State M. A. 13:385–390, 1916.
13. Robinson, V.: An Essay on Hasheesh, M. Rev. of Rev., pp. 66–68, 1912.

14. Medico-Historical Bull. *1*:7. 1920.
15. Ann. M. Hist. *1*:223–224, 1917.
16. Tjomsland, A.: Victor Robinson. *In* Kagan,[1] p. 439.
17. The birth of the bulletin, Medico-Historical Bull. *1*:2. 1920.
18. Sigerist, H. E.: A Tribute to Victor Robinson. *In* Kagan,[1] p. xi.
19. Robinson, V.: Pathfinders in Medicine, ed. 2, New York, Medical Life Press, 1929, p. viii.
20. Robinson, V.: The Story of Medicine, revised and enlarged edition, New York, New Home Library, 1943, p. 442.
21. Robinson, V.: A medico-historical curriculum, M. Life *37*:619–650, 1930.
22. Viets, H. R.: Edward Clark Streeter (1874–1947), Bull. Hist. Med. *21*:843–845, 1947.

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Some Observations on the Epidemic of Asian Influenza in Jerusalem

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Israel was reached by the pandemic of Asian influenza in September, 1957, and by the middle of October, a large proportion of the population had been attacked. Laboratory confirmation was obtained for the first time on September 26 by the Institute of Virus Research of the Medical School of the Hebrew University in Jerusalem, which also serves as the Regional Influenza Research Center of the World Health Organization. The agent identified on that occasion was of the Asian influenza–Singapore type 1957. The findings were confirmed by the Central Laboratory for Virus Research in London.

When the epidemic was in full swing, physicians were so overburdened that they could not be expected to conduct proper registration of their cases. Although the Ministry of Health requested notification, during those hectic days physicians just could not get around to reporting or even listing their cases. It was, therefore, evident from the start that any statistics based on notifications would be extremely incomplete and could not even be accepted as a basis for any kind of estimate.

The situation was complicated by the fact that the epidemic coincided with the High Holidays, when school attendance is irregular.

When school started properly after the Holidays, a large number of the students had already had the disease and had recovered. This suggested an opportunity of using the school children for a retrospective survey regarding the Jerusalem population at large.

A form was worked out in consultation with the Hadassah Department of Child and Student Health Supervision, the Department of Epidemiology of the Ministry of Health, and the Central Bureau of Statistics. The plan was to conduct some sort of quick sample survey, with retrograde information, which had to be compiled with utmost speed, as the success depended largely on the children's memory. It was, therefore, also impossible to work out a proper sample, but care was taken that schools from all parts of the city be included. In addition, it was decided to use for questioning only students of a certain age.

THE QUESTIONNAIRE

Following are the items included in the questionnaire: name; school; age; address; has child already had Asian influenza? if so, day of onset; how many days was he absent from school? how many children up to 18 years are there in the family (excluding the pupil himself)? how many of them have had the disease? how many

TABLE I

Percentage Distribution of Cases by Date of Onset

District		Total		September			October								
		No.	%	-25	26-28	29-1	2-4	5-7	8-10	11-13	14-16	17-19	20-22	23-25	26-28	29
1-34	All districts	536	100.0	3.0	2.6	3.7	6.2	8.9	23.3	14.0	11.0	11.2	8.8	6.3	0.6	0.4
1-2	Bucharim, Beith Israel	15														
3-5	Meah Shearim, Geulah, Kerem Avraham	47	100.0	—	—	8.5	4.3	6.4	21.3	8.5	19.1	10.6	10.6	6.4	—	4.3
6-7	Sichron Moshe, Mekor Baruch	52	100.0	—	3.8	—	3.8	1.9	30.7	17.3	13.5	19.2	3.8	5.8	—	—
8-9	Town Centre, Nachlat Shivah	68	100.0	1.5	2.9	4.4	5.9	4.4	19.1	10.3	13.2	14.7	10.3	10.3	2.9	—
10-11	Machane Yehudah, Sichron Yosef	33														
12-15	Nachlat Zion, Miskeret Moshe, Nachlat Achim, Shaare Chesed	64	100.0	1.6	3.1	3.1	3.1	17.2	29.7	14.1	9.4	1.6	12.5	3.1	1.6	—
10-15		97	100.0	1.0	3.1	2.1	7.2	18.6	22.7	12.4	6.2	9.3	13.4	3.1	1.0	—
16-18	Rehavia, Kiriat Shmuel, Merchavia	68	100.0	1.5	1.5	1.5	10.3	4.4	17.6	19.1	16.2	10.3	11.8	5.9	—	—
19	Talpioth	17														
24-25	Beth Hakerem, Kiriat Moshe	22														
29	Merkaz Mischari	15														
30	Romema	15														
31	Katamon	58	100.0	1.7	5.2	3.4	1.7	15.5	29.3	19.0	10.3	6.9	1.7	5.2	—	—
33-34	German colony, Bakaa	41	100.0	17.1	2.4	12.2	4.9	4.9	24.4	17.1	4.9	4.9	7.3	—	—	—
	All others	21														

Percentage calculations have been made only for figures where the total for the particular district or group of districts was more than 40.

adults (older than 18 years) are there in the family? how many of them have had the disease? how many rooms are there in the apartment?

The survey was made during three days (October 27 to 29). Approximately 1000 forms were distributed but only 743 of them were completed in a way suitable for statistical treatment. The average age of the children questioned was 13 years in secondary schools, 15 years in high schools. A short explanation of how to complete the questionnaire was given by the author or by a school nurse. Forms were then collected and forwarded to the Health Section of the Central Bureau of Statistics for statistical treatment.

The statistical tabulations were based on the areas of the city where the students lived without distinction between the individual schools. This was done in order to get an idea of the spread of the epidemic in the city. The inquiry covered 4045 persons, 2133 of whom were less than and 1912 more than 18 years of age.

Table I shows the percentage distribution of cases in the period from Sept. 29 to Oct. 29, 1957. Table II gives the attack rate (up to October 29) in the different areas of the city in two age groups: (a) school age—up to 18 years—and (b) age 18 years and older. This gives an indication of the spread of the epidemic in the different socioeconomic strata of the population. Table III indicates the correlation between the attack rate in the family and the degree of crowding. Table IV shows correlation between period of illness and degree of crowding.

The city of Jerusalem falls into 32 districts, which, for the sake of clarity, have been summed up to 15 somewhat larger units. In addition, in the tables the totals are given for districts 1 to 5, which have a not particularly well-to-do population, and for 10 to 15, which are the least fortunate and most crowded of the city. It seems worthy of note that in the Jerusalem population, differences in income are not too great.

COMMENT

It is shown in table I that in the period September 25 to October 1, the first cases appeared in the German colony and Bakaa. Although the peak for these areas was the same as for the others (October 8 to 10), the epidemic evidently started there and then spread to the others. Peak frequencies continued for about 10 days, and by the end of the month numbers were again low.

In table II originally the two types of schools (elementary and secondary) had been treated separately. However, as it became apparent that there was no particular difference between the two, I included only the total for both.

From table II it appears that approximately 67 per cent of the population under survey up to the age of 18 years, and 38 per cent of those older than 18, were attacked in the period from Sept. 29 to Oct. 29, 1957. For all ages together, the attack rate was 53 per cent. It is worthy of note that in the more well-to-do districts of the city (16 to 18) the attack rate was, for age less than 18 years, only 60 per cent and for all ages, 43 per cent, and in the even wealthier areas (25 and 26), 56 and 38 per cent, respectively. On the other hand, in the less developed districts (1 to 5) the respective rates were 71 and 56 per cent, respectively, while for districts 10 to 15, known to be greatly overcrowded, the respective rates were 70 and 61 per cent.

TABLE II

Percentage Attack Rate by District of City

	District	Absolute no. of persons attacked	Per cent of Attack						
			All schools			Secondary schools		Elementary schools	
			All ages	<18	>18	All ages	<18	All ages	<18
1–34	All districts	2,150	53	67	38	52	68	54	67
1–5		259	56	71	43	44	60	62	76
1–2	Bucharim, Beith Israel	77	64	71	56	62	60	64	76
3–5	Meah Shearim, Geulah, Kerem Avraham	182	53	70	38	37	60	61	77
6–7	Sichron Moshe, Mekor Baruch	173	47	64	32	42	60	49	65
8–9	Town Centre, Nachlat Shivah	217	47	65	30	46	63	48	66
10–11	Machane Yehuda, Sichron Yosef	179	65	76	51	57	63	66	77
12–15	Nachlat Zion, Miskeret Moshe, Nachlat Achim, Sha'are Chesed	318	60	67	50	58	63	60	66
10–15		497	61	70	50	57	70	62	70
16–18	Rehavia, Kiriat Shmuel, Merchavia	201	43	60	25	44	65	42	59
19	Talpioth	68	54	63	25	64	65	47	54
24–25	Beth Hakerem, Kiriat Moshe	72	38	56	24	45	65	35	57
29	Merkaz Mischari	50	39	59	19	50	65	37	58
30	Romema	76	60	71	46	60	65	60	74
31	Katamon	293	64	74	50	67	82	63	72
33–34	German colony, Bakaa	165	55	70	20	57	82	54	70
	All others	66	55	70	20	57	82	54	70
	Not stated	13	55	70	20	57	82	54	70

Table III shows that where there were less than 2 persons/room, the attack rate within the family was 41 per cent; with 2 to 3 persons/room, it was 60 per cent; where there were more than 4 persons, it was 70 per cent. This is in agreement with other epidemiological observations indicating a positive correlation between crowding and the spread of epidemics.

In addition, it is seen from table IV that where the degree of crowding was higher, periods of absence from school were shorter—a phenomenon already observed in this country in connection with other diseases.

The object of the survey was to obtain a certain kind of information with a speed that did not allow the planning of a proper sample. Nevertheless, as has already been pointed out, care was taken to select schools for investigation all over the city in order to make the material as representative of the population at large as possible. In almost every family investigated, more than 1 person was attacked. It should, however, be taken into account that it may now and then have happened that the illness reported as Asian influenza might have been an ordinary upper respiratory

TABLE III

Correlation Between Room Index (Number of Persons/Room) and Per Cent Attack Rate in Family

Room index	Per cent attack rate							
	Total	0–20	21–40	41–60	61–80	81–100	Mean	Median
Total	735	158	145	146	147	139	49.0	48.8
0.0–1.0	93	26	28	15	15	9	39.9	34.6
1.1–2.0	340	89	69	83	48	51	44.3	42.9
2.1–3.0	148	29	29	19	37	34	52.4	56.8
3.1–4.0	79	7	12	18	23	19	58.9	62.2
4.1–5.0	37	3	3	5	11	15	67.3	73.6
5.1–6.0	12	2	1	2	4	3	58.3	65.0
6.1–7.0	7	1	—	2	2	2	61.4	65.0
7.1–8.0	4	1	—	—	2	1	60.0	70.0
8.1–9.0	10	—	2	1	3	4	68.0	73.3
9.1–10.0	2	—	—	—	1	1	80.0	80.0
10.1	3	—	1	1	1	—	50.0	50.0
0.0–2.0	433	115	97	98	63	60	43.3	40.9
2.1–4.0	227	36	41	37	60	53	54.7	59.7
4.1	75	7	7	11	24	26	64.7	70.4

infection. In addition, the figures for date of onset and period of illness should be accepted with a certain reservation and a deviation of a few days in either direction should not be excluded.

Regarding the number of persons living in a family, it was explained to the students that this should include only those actually living in the apartment in the period under survey, and it is not very probable that the children made mistakes regarding this point. On the other hand, mistakes regarding the number of rooms at the family's disposal are more probable, but overstatements are not likely to have affected the correlation between the attack rate and crowding.

The bias created by the fact that the population investigated consisted exclusively of families with children is more serious. The calculations given here show that the tendency to contract the disease is greater in school children than among adults. This, together with the fact that the school child is an effective go-between for the spread of an epidemic, makes it likely that in families with school children the attack rate would be higher. On the other hand, during an epidemic of that size, anyone who has contact with other persons may get the virus. It may, therefore, be assumed that although the general attack rate among adults might have been lower than that observed in our survey—and this refers especially to elderly persons who no longer live with children—it was not so to an appreciable degree. It seems, therefore, permissible to make an estimate, although a very rough one, of a general attack rate for the whole population of the country of no lower than about 40 per

TABLE IV

*Correlation Between Room Index (Number of Persons/Room) and Days of Illness (Absence from School)**

Room index	Total no. days absent	No. days of absence from school											Mean	Median
		1	2	3	4	5	6	7	8	9	10	11		
Total	483	13	22	45	76	75	68	88	43	13	19	21	5.7	5.7
10.1+	2	—	1	—	—	—	—	1	—	—	—	—	4.5	4.5
9.1–10.0	2	—	1	—	—	—	—	—	—	—	—	1	6.5	6.5
8.1–9.0	10	—	1	2	2	2	1	—	—	—	—	2	5.4	4.5
7.1–8.0	2	—	—	—	—	—	2	—	—	—	—	—	6.0	6.0
6.1–7.0	5	1	—	1	1	1	—	—	—	—	1	—	4.6	4.0
5.1–6.0	8	—	2	—	4	—	2	—	—	—	—	—	4.0	4.0
4.1–5.0	22	2	5	6	6	2	2	1	3	—	1	1	4.4	3.8
3.1–4.0	55	—	2	8	12	12	6	6	—	3	4	2	5.5	5.0
2.1–3.0	102	1	4	15	19	12	12	19	10	3	1	6	5.7	5.5
1.1–2.0	217	9	5	12	29	37	36	46	22	6	7	8	5.9	6.0
0.0–1.0	51	—	1	1	3	9	7	15	8	1	5	1	6.7	6.7
4.1	58	3	10	9	13	5	7	2	3	—	2	4	4.7	4.0
2.1–4.0	157	1	6	23	31	24	18	25	10	6	5	8	5.6	5.2
0.0–2.0	268	9	6	13	32	46	43	61	30	7	12	9	6.1	6.2

* Including students who had not been attacked at all.

cent and no higher than about 60 per cent. Consequently, the number of patients, in a population of 2 million, can be estimated to be between 800,000 and 1,200,000.

SUMMARY

A quick survey was made in school children of the higher forms regarding the spread of the epidemic of Asian influenza in Jerusalem. The results of this survey show: (a) that the epidemic started in Jerusalem late in September, 1957, apparently spreading from the Bakaa Quarter and the German colony; (b) that the peak was observed between October 8 and 10 but numbers remained high for 10 days; (c) that toward the end of October cases were already few; (d) that 79 per cent of the students questioned had been attacked, while of all the children up to 18 years in the family circle included in the investigation, 67 per cent contracted the infection; in the entire population included in the survey (4045 persons), the attack rate was 53 per cent; (e) that there was a clear correlation between attack rate within the family and degree of crowding; (f) that there was a correlation between low number of days absent from school and degree of crowding.

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Radioactive Sodium Aids Bone Surgeon's Diagnosis

Early diagnosis of death of the top of the thigh bone, resulting when its blood supply is cut off, is now possible, two researchers reported in London recently.

By injecting sodium-24 into the neck of the femur, the surgeon can immediately diagnose whether or not the blood supply to the bone is being maintained. If it is not, said Drs. P. G. Laing and Albert B. Ferguson, Jr., of the University of Pittsburgh's department of orthopedic surgery, steps can be taken to treat the condition immediately.

In cases of fractures of the thigh bone neck or dislocations of the hip, the patient is thus spared a second operation, the scientists pointed out in the British scientific journal *Nature* (November 22).

Experiments with large dogs showed that when the femoral head was devascularized and injected with the sodium-24, radioactivity counts fell by only 5 per cent in 10 minutes. However, when the blood vessels were left intact, the counts obtained with a scintillation tube fell by 20 to 50 per cent in 10 minutes, the scientists reported.

The Epic of Medicine*

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It is now two years since we undertook the quixotic task of integrating within the pages of the *Newsmagazine MD* the three spheres of life—professional, social, and human—and the three personalities—physician, citizen, and man—of the physician, and thus attempted to help him to attain that plenitude which is attained only when man renounces to nothing in life. Thanks to our colleagues' constant support and encouragement, *MD*, more than a magazine, is today a magic carpet on which the physician may soar through the blue skies of the spirit.

Our only compass has been our desire to make *living* medical history by looking with eyes filled with love and wonder at the world and age in which we live, our place in space and our point in time, and by applying to both minor and major events in Medicine and in life the delicate instrument of History—History, without which nothing has any real meaning. For only History can help man to understand the present and improve the future.

These have been two years of unremitting effort and rewarding harvest. Each month we have erected a complex editorial scaffolding from which, with words and pictures and a drop of common sense—the least common of the senses—we have tried to paint a mural of the ideas, men, events, and things of the world we live in, a mural true and realistic, but also warm and colorful, for we prefer the fresh flowers that gracefully lift their perfumed heads in a garden to the faded pressed flowers in a rare herbarium.

Like a mountaineer on the ascent, issue by issue, *MD* has broadened its range of vision. We have also sought to dig deeper into the core of things and the essence of man. In enveloping the physician's work in the rich fabric of History, we have turned History—which is remembrance—into a dynamic instrument for the conquest of the future—which is hope.

A magazine's life, like that of a human being, must move forward, must continuously project toward the future, must keep on making new plans. Hence, in this third year of its life, *MD* will launch a new project, inspired by my forthcoming book *The Fabric of Medicine*, a monumental triptych on the History of Medicine, consisting of *The Epic of Medicine*, *The Physician's Saga*, and *The Patient's Progress*, which for the first time will present the History of Medicine dramatized by means of all the resources offered by the modern graphic arts. Thus we shall open a new picture window not on space but on time.

History is above all pageantry. With all the pomp and glory of pageantry, *The Epic of Medicine* will present the History of Medicine as craft, art, and science, relating it to the history of civilization, of the arts, and of science and technology.

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* This editorial originally appeared in *MD* Medical Newsmagazine, January 1959.

A series of living tapestries will portray the joys and miseries of physicians throughout History. Colored *tableaux vivants*, romantic yet realistic, will revive great moments in medical history and the lives of great medical figures. Men, ideas, events, and places of memorable import in the story of Medicine will be resurrected. Like multicolored lamps, words and pictures will light up the still dark corners of the healing art. Like rainbow lamps, they will conjure a great festival of the medical spirit in the multisplendored garden of History.

Pen, brush, and camera will take us on a safari across medical history's wonderland. They will show us the great medical shrines and landmarks that the centuries have failed to destroy: Hippocrates' plane tree on the sunny isle of Cos, the ancient walls of Byzantium, Avicenna's tomb in Persia, Boerhaave's house, the golden vineyards where Claude Bernard lived; and they will reconstruct for us in all their splendor those landmarks that have long been gone, thus uniting past and present and restoring flesh and soul to what, in our mind's eye, are only things of mist and smoke.

The Epic of Medicine will relate the first awakening of medical consciousness in the magic-governed shamans of the primitive world; the fight between sorcerer-physicians and the demons of disease in Mesopotamia and Egypt; the ceaseless spinning of subtle philosophies by philosopher-physicians amidst the harmonious geometry of white marbles in ancient Greece; the practice of Medicine by slave-physicians in bureaucratic, militaristic Imperial Rome; the compilation of monumental medical works by physicians in walled-in Byzantium, who thus sought to escape their cloistered world, just as the Byzantine artist sought escape through the magic windows of multicolored miniatures and the stained glass windows of basilicas; the achievements in alchemy and hygiene of the Arabian *hakims* in the Baghdad-to-Cordova empire created at the point of scimitars by Islam; the birth in the so-called "Dark" Ages of the first hospitals and universities, plus the Gothic cathedrals and *The Divine Comedy*, which made this an era not of darkness but of blazing splendor; the exploration of the human body by artist-physicians in the Renaissance, which paralleled the exploration of the new world beyond the Atlantic waters; the discovery of the circulation of the blood and the beginning of scientific research in the Baroque, a period characterized by motion and emotion in art; the endeavors of the experimenters and visionaries of the Enlightenment and the Romantic periods, who heralded the transition to last century's naturalist positivistic medicine; the transition from medicine based on qualitative impressions to present-day medicine, based on quantitative measurements, which has led to psychiatry, antibiotics, ataraxics, and space medicine, and is leading to a biochemical, physical, and—once again—philosophical medicine and to the submission of man and the universe to objective evaluation and subjective integration.

The Epic of Medicine will consist of twelve installments and will be followed by *The Physician's Saga*, a series depicting the story of the physician as an individual, exemplified by the lives of great physicians, and *The Patient's Progress*, which will be dedicated to the forgotten and yet the most important man in the History of Medicine: the patient.

The Epic of Medicine will endeavor to give substance and meaning to our daily work. For, the History of Medicine *is* Medicine. Our medical activities—administering an antibiotic, making a psychoanalysis, performing a laparotomy—acquire meaning

only when we interpret them in reverse, like a film shown backwards. Only thus can we find the historical meaning in everything we do in our profession. Our medical work—preparing a clinical history, making a diagnosis, applying treatment—would be far more efficient if we knew the *why* of what we are doing and if we could anticipate its future course. This is possible only by making, as we shall try to do in this series, *living* history of all medical work, and by doing this with love and imagination, so as to let fly once again the many-hued butterflies pinned between the pages of the huge book of History.

There are three ways of handling historical events: if they are minor, they can be *described;* if they have historical dimension, they can be *interpreted;* if they can be integrated with other events in life, they can be *narrated* like a true tale of wonder. This *MD* intends to do. There are many *histories* of Medicine, but there is only one *History* of Medicine that is related to the history of civilization. Through the magic of interpretative narration, to turn all the stories of physicians, patients, countries, and epochs into *one* narrative, at once living and practical, imaginative and realistic—this is the new thread we seek to weave into the fabric of our dream.

SYMPOSIUM ON MEDICINE AND WRITING

The Symposium on Medicine and Writing that appeared in the November 1956 issue of INTERNATIONAL RECORD OF MEDICINE has been published recently as a Monograph. The articles included in this Monograph are: "The Editing of a Modern Medical Textbook" by Russell L. Cecil; "Plain Talk and Clear Writing" by Morris Fishbein; "The Principles of Bibliographic Citation" by John F. Fulton; "The Art of Communication" by Joseph Garland; "On Writing a History of Medicine" by Douglas Guthrie; and "Minerva and Aesculapius: The Physician as Writer" by Félix Martí-Ibáñez.

This 72-page Monograph is sold for $3.00. As the fourth in the series of MD International Symposia, this book is the companion piece of *Medical Writing*, which was published in May 1956.

To obtain this monograph, write to MD Publications, Inc., 30 East 60th Street, New York 22, N. Y.

MD MONOGRAPHS ON MEDICAL HISTORY NO. 2

A History of Ophthalmology

by GEORGE E. ARRINGTON, JR., M.D.
Associate in Ophthalmology
Medical College of Virginia, Richmond, Virginia

Foreword by FELIX MARTI-IBAÑEZ, M.D.

A History of Ophthalmology presents a study of the development of the "ophthalmological mind" throughout the history of civilization. The book attempts to guide the ophthalmologist to the sources of concepts that regulate his daily clinical functioning. He is introduced to the great thinkers who established, through both reasoning and experimentation, the basic ideas of visual function and dysfunction on which the science of ophthalmology is founded. Ophthalmology is carefully related to medicine generally in this work, and their roles of interdependence and interaction are shown. 192 pp., clothbound, price $4.00.

A History of Ophthalmology is the second book in a series written and designed for physicians, sociologists, educators, and members of related professions, as well as residents, interns, and students. Under the editorial direction of Félix Martí Ibáñez, M.D., Professor and Director of the Department of the History of Medicine, New York Medical College, Flower and Fifth Avenue Hospitals, the series is designed to enrich the physician's knowledge of medicine and to impart useful information. *A History of Public Health* by George Rosen, M.D., Ph.D., M.P.H., was recently published. Soon to appear is *A History of Neurology* by Walther Riese, M.D. Beautifully designed and attractively bound in durable cloth, *A History of Ophthalmology* exemplifies the ultimate elegance in the art of bookmaking and will make a handsome addition to your library.

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